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INDUSTRIAL MEDICINE AND SURGERY

DECEMBER, 1958

Industrial Thallium Intoxication

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Ohio Department of Health

Thallium has been recognized as a toxic element since soon after its discovery in 1861, for the man who isolated the metal suffered from its effects.

An extensive search of the literature available prior to 1934 revealed 778 cases of human toxication with 46 deaths.[1] Six hundred ninety-two of the cases and 31 of the deaths resulted from clinical use of thallium salts.

Thallium acetate was used to reduce night sweats in phthisis at the end of the last century; daily doses as low as 5-10 mg. per day produced symptoms.[2] For a short time it was also tried for the treatment of diarrhea. The most widespread medicinal use of thallium was as a dipilatory in the treatment of tinea of the scalp. As a rule, children tolerated fairly well a single dose of 8 mg. of thallium acetate per Kg. Even at this dose, of the 3,648 treated,[1] six children died and 216 were poisoned; two under-nourished, preteen boys died following treatment with a total of 5.5 mg. per Kg. given in divided doses. Teleky, as reported by Carozzi,[3] had previously suggested that repeated small doses may be more apt to cause permanent injury than a single dose.

During the late 1920's a dipilatory containing about 7% thallium sulfate appeared on the market. Its popularity was short-lived. Cases of poisoning soon appeared, many of which were diagnostic problems. One notable incident reported was the admission to a single hospital of three women whose chief complaint was loss of vision.[4] They were suspected of having brain tumors, and thallium intoxication was not considered until the second patient volunteered the information that she had been using this dipilatory cream. Further questioning of the first and third patients revealed that they, too, had used it. However, Sabouraud[5] had reported that a cream containing 1% thallium had been used for 18 months without ill effects when used on limited areas of the body and in amounts not to exceed the size of two kernels of wheat each day.

No deaths were reported by Munch in 1934 from use of the metal through skin absorption of dipilatory creams or from industrial exposure. Of the 12 known industrial intoxications found in three German plants, nine persons recovered following termination of exposure while two had residual chronic albuminuria and one had permanent blindness. Five Prussian plants handling thallium, five German chemical manufacturers

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using the metal on a commercial scale, and two American dealers reported no evidence of intoxication among their personnel. The total number of exposed people was not given. These same 12 workers had been reported on by different authors and evaluation of the data was difficult.[1]

In one of the three plants where intoxication occurred, the men were engaged in the recovery of thallium from flue dust in a sulfuric acid works.[6] All had lymphocytosis and eosinophilia. Permanent blindness occurred in one young man after only three months exposure.

Heyroth[7] reviewed the literature in 1947 and reported no new cases of industrial poisoning. However, in referring to the cases of Teleky and those of Rube and Hendricks, he states that most of the workers engaged in producing the metal or its salts were affected.

The next reported cases of industrial thallium poisoning in two rodenticide workers appeared in the Swedish literature in 1951. The onset in one simulated a heart attack.[8] In 1955, four additional cases were found in four different Swedish plants engaged in the manufacture of rodenticides. The patients had been careless in handling the powder and did not suspect poisoning. Sensory changes in the distal part of the limbs were accompanied only by minor and uncertain signs of polyneuritis. Only one had demonstrable signs of alopecia.[9]

At about this same time there was reported in Germany, illness in a 45 year old lithopane worker after 24 years of working with thallium. He had worn protective clothing and a mask; but despite this, symptoms of ascending polyneuritis of the Landry type, disturbances of speech and sight, and hair loss appeared. Although he improved under treatment, he was still incapacitated after eight months.[10]

A search of the literature does not reveal any cases of intoxication due to thallium in American industry.

The Ohio Study

Our attention was directed to this particular industrial operation as a result of a request from a physician seeking aid in the diagnosis of a case of suspected thallium intoxication (Employee number 14).

Description of Process and Exposure: The plant where this man was employed is engaged in the separation of industrial diamonds from abrasives. One step in the process depends on differential separation of the diamonds from certain minerals by gravity in varying concentrations of solutions of organic thallium salts.

The original plant burned in January, 1957, and the company had been housed in its present quarters approximately 10 months at the time the investigation was made.

All operations involving the use of thallium were confined to a corner room approximately 15 by 18 by 8 feet. Casement-type windows were on the north and west walls. A door on the south wall led to the room where initial chemical treatment of the dust was performed. To the east was the washroom and office.

The men employed in the thallium extraction worked alone; one being assigned to each of two shifts. Reconcentration of the thallium salts solution was effected by distillation in a closed system under a hood supplied with an exhaust fan. Gravitation was carried out in centrifuge cups, the outsides of which contained dried salts left on the surface when supernatant fluid and the lighter minerals were decanted. Handling of these, and spillage, caused the workers' hands to acquire white stains which could not be removed by simple washing.

The first man to be employed in this process started working October 1, 1950. Between then and February 14, 1958, when the process was discontinued, 15 men had been employed to work in the operation.

Materials and Methods: To determine the extent of the problem encountered in using thallium in this plant, it was decided to study the work and medical histories of the men employed in the operation. All 15 men, and relatives of 12 of them, were interviewed. Where possible, family contacts were made separately.

The physicians attending these men during or immediately after their employment were contacted for pertinent medical data. Where applicable, hospital records and Industrial Commission files were reviewed.

Blood pressure readings and laboratory tests were done on some of the men on a selective basis. Field methods for testing were utilized wherever the time element was factor in giving accurate results.

The blood was diluted with Hayem's solution for red cell count, and 0.1N HCl for white count and hemoglobin determination in a Hayden-Hauser Hemometer. Blood smears for differential and stipple cell count were returned to the laboratory and stained with Wright's stain.

The urine was tested with Testape from Eli Lilly and Company, and Ictotest, Bumintest, and Hematest from Ames Company. Porphyrins were determined by modification of de Langen and ten Berg[11] method: 3 cu. ml. urine was acidified with four drops 6N acetic acid and thoroughly shaken with 3 cu. ml. ether. These tubes were left under cover for 20-30 minutes and read for fluorescence under a Blak-ray long wave ultraviolet light. Reaction was determined with pHydrion paper.

All urine specimens were collected in acid-rinsed containers and returned to the laboratory

for thallium determinations. When the urine was positive for thallium, repeat specimens were obtained for chemical analysis.*

On February 13, 1958, an industrial hygiene engineer collected samples for atmospheric thallium concentrations in the work room.

The following are case histories of some of the men who had several symptoms of thallium intoxication prior to the time of this investigation. The last four cases were diagnosed by the employees' personal physicians and claims alleging thallium intoxication were approved for compensation by the Industrial Commission of Ohio.

Employee number One was employed from October 1, 1950 to January 31, 1954; since then he has been a painter. This 22 year old male was admitted to the hospital October 28, 1950, about one week after the onset of a dull ache and pain in the epigastrium, constipation, and chest pain on taking a deep breath. He had had some relief for about one-half hour with Alka-Seltzer, but none with food. He also complained of extreme fatigue of the legs.

Examination revealed generalized abdominal tenderness, most marked in the epigastrium. GI and GB series were within normal limits. X-rays of the feet showed minimal hypertrophic changes involving some of the tarsal joints of both feet.

On October 29, 1950, urinalysis was negative and blood count was RBC 5.15 M; Hb 14.5; lymphs 38; WBC 7,250; segs 58; eos 1; stabs 3; color index 1. On October 31, 1950, gastric analysis showed no free acid and total HCl was 14, 10, and 10 for fasting, first and second hours. Serum amylase was 95 mgm. percent on November 6, 1950. Blood pressure ranged from 150-180 systolic and 108-120 diastolic during his hospital stay.

An appendectomy was performed on November seventh, and five days later he was discharged from the hospital on a gastritis diet and HCl five drops twice daily. The pathologist reported a mild acute catarrhal appendicitis but that the symptoms may have resulted from the long, kinked course of the appendix.

The patient states that his hair started coming out while he was in the hospital, and although he subsequently had some regrowth, still has far less than his older brothers.

On November 17, 1950, ten days following his appendectomy, his blood pressure was 120/70. He returned to work early in December and on January 2, 1951, he weighed 131 pounds and had a blood pressure of 130/80.

During the next four years he had daily exposure to thallium but did not always work at this job for the full eight hours. He continued to have trouble with his stomach, had difficulty sleeping and was nervous and irritable.

In May, 1953, he was turned down for insurance and consulted his private physician. He weighed 169 pounds and had a blood pressure of 170/90, and rales in both bases. His chief complaints were abdominal pain and rectal bleeding. Chest x-ray was normal. Results of GI series were comparable with the one done in 1950. Barium enema revealed a slightly contracted caecum with a minimum of irregularities.

On December 31, 1954, he again visited his physician. His chief complaints were occasional vertigo and tinnitus. He denied having abdominal pain except after eating fried foods. He now weighed 172 pounds and had a blood pressure of 174/110 and was put on Raudixin; 50 mg. twice daily.

He did not see a physician again until after his interview regarding this investigation. He has had no rectal bleeding, and now eats everything, although he still has some trouble with his stomach. He gets a tight feeling across his chest at night and has ringing in his ears. Blood pressure taken in March ranged from 150/90 to 180/110. He was placed on Reserpine; 0.25 mg. every other night. In June, 1958, his blood pressure was 150/90.

This patient states that at age 18, while serving with the Army in Italy, he had a physical examination. After having his blood pressure taken, he was told it was a little high and told to rest. A recheck in a few minutes was reportedly within normal limits. His mother, who is now deceased, had high blood pressure, as do two, and possibly all, of his three maternal aunts.

A communication in 1958 from the physician who treated this man from 1950 to 1954 stated that he feels this man had thallium intoxication.

Employee number Two was employed from October 1, 1951, to January 2, 1952, and part time from June, 1951, to September, 1951. Previous employment had been as a paint sprayer for 15 years. This 34 year old man was admitted to the hospital November 13, 1951, with a chief complaint of an abdominal pain of 10 day's duration; also pain in shoulder regions and a tight feeling across the epigastrium. The pain became severe and several soft stools were noted; T. 99, B/P 120/70; temperature 98.8° at 6 p.m. and 99.4° later in the evening. The pain was over the entire right side with as much pain over McBurney's point as in RUQ with moderate right-sided rigidity and rebound. The patient had had a similar episode in 1947, at which time all GI and GB series were negative and a diagnosis of chronic appendicitis was made. WBC was 14,500 with segs 60, stabs 6, eos 1, lymphs 32, and monos 1. His appendix

*A report of the methods used in determination of thallium content of urine and other biological materials will be prepared at a later date by Mr. Edward Borawski, Chemist, Division of Industrial Hygiene, Ohio Department of Health.

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was removed November 13, 1951, with no evidence of recent acute inflammation reported. Additional laboratory work was performed on November 17, 1951: RBC 3.96, WBC 8,700, Hb 11.5, color index 1, stabs 10, segs 65, lymphs 25. No abnormalities were reported in the urine. On November 20, 1951, sed rate was 19 mm, PCV 47, and urine was negative for porphyrins. He was discharged November 21, 1951 with numerous complaints of pain in the abdomen and legs.

He was readmitted to the hospital on January 2, 1952 with nausea, vomiting once, abdominal pain and generalized aches and pains which had been present and getting worse since his appendectomy on November 13, 1951. Abdominal pains were crampy in nature and radiated to the back. The joints seem to be involved, but no tenderness or swelling had been noted. Because of persistence of the symptoms the patient had become very nervous and irritable. He was apprehensive and apparently acutely ill. His temperature upon admittance was 98.6°, with findings limited to voluntary rigidity and exquisite tenderness of entire abdomen and stasis eczema of the right ankle. Diagnoses considered were peripheral neuritis of undetermined etiology, collagen disease and multiple myeloma.

On medical consultation no evidence was found of polyneuritis as evidenced by complete abolition of tenderness when the patient's mind was otherwise occupied and tenderness was not of nerve distribution type. He was extremely tense during examination and perspired profusely. Resultant impressions were severe anxiety state and possible thallium poisoning.

Periodic differential counts revealed 7% eosinophiles, and 16% stabs on January 10, 1952, and no eosinophiles, and 26% stabs on January 12, 1952. A trace of albumin was found in the urine on January 3, 1952. A faint trace had been detected during a hospitalization in August, 1951.

A urine sample collected January 12, 1952, contained 0.03 mg. lead per liter, less than 0.02 mg. As., and 0.008 mg. Hg. per liter. A qualitative test for thallium was positive.

GB and GI series were normal and x-rays of the lumbosacral spine showed no evidence of disease of bones or joints. The pathologist reexamined the appendix and found in a large number of slides only two instances of any arterial changes. The pathologist reported, "One of these was very striking and consisted of a marked necrosis of the wall, fibrinoid degeneration and infiltration by large numbers of histiocytic cells. Other small arterioles likewise were seen which show some hyalinization and possibly may represent a long-standing healed reaction. Diagnosis; arteriolitis, necrotic, acute and chronic."

The patient was treated with BAL 180 mg. every six hours for two days then once daily from January 5 to 16, 1952, and with vitamin B_{12}, 30 mg. daily from January 8 to 16, 1952. No improvement was noted by January 9, 1952. Blood calcium was 13.3 mg. % on January 5, 1952, and a diagnosis of hyperparathyroidism was considered. Blood calcium and phosphorus were repeated January 11, 1952, with findings of 11.89 and 2.95 mg. % respectively.

On January 12, 1952, the spinal fluid contained 83 mg. % sugar, 74 mg. % protein and 114.9 m.e.q. chlorides. Gold curve was 1122211000 and Wasserman was negative. The Thorn test done on January 14, 1952, showed adequate adrenal function.

Employee number Five was exposed from June 8, 1952, to August 30, 1952; all of his previous and subsequent employment was out-of-doors. This 24 year old man was first ill on August 28, 1952, with nausea, vomiting, constipation, abdominal cramps and pains and numbness of both arms and legs. According to the patient, his physician (same one who saw Employee number One) first made a diagnosis of appendicitis until he learned that these two patients had been similarly exposed. The patient was treated at home with enemas and hot baths. He lost over 30 pounds. While in bed his hands and legs "drew up," he had difficulty thinking and was depressed and acted "lost" for several months after his illness, and he continued to have abdominal distress for about two months. He does not feel that his sense of touch is as good as it used to be. About December, 1952, he was told he had albumin in his urine.

Sometime during his employment a urine sample was collected at work and analyzed at a private laboratory. The company advised the Industrial Commission of the results of this analysis as 0.57 mg. thallium per liter of urine.

Employee number 11 was employed in the thallium operation April 22, 1957, to July 22, 1957. His previous employment included one year as millwright in a steel mill and 13 years in cotton mill. About the middle of June, following a sunburn, this 30 year old man noticed the first symptoms and was absent from work June 17 and 18. He had cramps on inner aspects of his thighs and ankles, especially on arising in the morning. He first visited a physician July 24, complaining of a numb feeling in the legs starting below the knees and extending down over the dorsum of the feet and middle three toes. For two weeks he had had nausea at the sight of food and epigastric pain starting about an hour after eating and lasting for about an hour. He had lost about 14 pounds and

complained of easy fatigability. There were no apparent hair changes, eye symptoms or signs of optic neuritis. On July twenty-fifth RBC was 5.77, Hb 14.8, WBC 5,300, neut 55, stabs 4, eos 3 and lymphs 38. Platelets were normal. On September 2, 1957, 17 grams of blood and 92 ml. of urine were sent to a private laboratory for thallium determination. No thallium was found in the blood and the urine contained 0.21 mg. thallium per liter.

At this time numbness was the only remaining symptom and the area involved and intensity of this was decreasing. Numbness gradually diminished, but some persisted for several months.

Employee number 14 was employed in the thallium process from December 14, 1957, until January 9, 1958, when, half an hour after beginning work, this 26 year old man went home because of illness. He reported he had been very tired and had aches and pains in his back and legs for several days. The day before he had abdominal pains and general weakness. His last previous occupational experience was drilling steel samples for laboratory analysis; this is a low chrome steel containing 0.0 to 0.8% Mn. and no lead. He had done this work two and one-half years, ending in October, 1957.

He was admitted to the hospital January 16, 1958, with the chief complaint of abdominal pains. Physical examination revealed blood pressure 160/90, slight congestion of the throat; heart rate regular with splitting of first sound, accentuation of second. $P_2 > A_2$ systolic murmur Grade I-II. He had a small ulcer of index finger of right hand where thallium solution had come in contact with a blister. He had slight-to-moderate numbness and mild weakness of both extremities. No pathological reflexes were elicited.

X-ray studies, including chest, abdomen, GB, GI, pyelogram and wrists and ankles, demonstrated no pathology.

On January twentieth eye examination was negative for any signs of thallium intoxication or any systemic disease. Three days later the neurological consultant found a very definite unsustained nystagmus of moderately coarse degree on right lateral gaze with some fine unsustained nystagmus on left lateral gaze. There was no other evidence of any other cranial nerve involvement.

Report of continued neurological examination disclosed no objective sensory disturbances in terms of pain, touch, position or vibratory sense. He gave a history of having for a few days an intermittent and lessening parasthesia over the plantar aspects of the feet. He had postural and positional vertigo lasting for about five days, beginning January 18, 1958. He had marked hair loss with shampooing, combing or mild traction. Weakness had progressed to the point where he was unable to walk. Motor strength was objectively normal and in the Romberg test there was slight sway but no true staxia. Conclusion of the examination was that he had definite, although mild, neurological manifestations of the heavy metal poisoning, but gave no evidence of involvement of the optic nerve and only mild evidence of a polyneuritic disturbance.

Hemogram on January sixteenth: Hb 13.5, WBC 6,800, stabs 1, segs 56, lymphs 38, eos 2, monos 3, hematocrit 41. Admission urine showed a trace of albumin; 3 plus on February tenth and 1 plus on February thirteenth, and negative on February fourteenth, fifteenth and sixteenth.

On January twentieth a thymol-turbidity test was 1.8; cephaloflocculation was 2 plus at 0-hour and 3 plus in 24 hours, and was negative on February tenth. Serum transaminase: 26 units on January twenty-fifth and 48 units on February tenth. Other laboratory tests on blood were essentially within normal limits.

This patient was treated with BAL for 12 days at 167 mg. intramuscularly every 6 hours for two days, then twice a day for 10 days. He was discharged February 16, 1958. Vitamins were started in January and continued through May, 1958. By May fifth, he had regained 13 pounds and was feeling and looking well.

Blood pressure on March 13, 1958, was 120/76.

Results

Of the 15 men employed in this operation, 12 had complaints which could have stemmed from their thallium exposure. The three who noted no physical change during their exposure, other than staining of their hands, were employed between June, 1955, and January, 1957. The fourth man who worked during this period experienced no other discomfort until after operations were resumed in April, 1957. All other workers, ranging in age from 20 to 41 developed their symptoms between three weeks and three months following their first exposure (Table I).

Nine were ill enough to seek medical care; the last man employed did not visit his physician until after the investigation had begun. Chemical analyses of urine samples of five of these patients indicated they had had exposure. Diagnosis of thallium intoxication was made in these five men, and considered in retrospect in a sixth. Heavy metal poisoning was suspected in still another employee, but the man was not working and did not feel financially able to undergo additional diagnostic tests.

It must be remembered that the records of the men not under medical care have no verifying dates, but are recorded as presented by the

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worker or his family from memory. This, in some cases, necessitated recall of events of five to six years past.

The individual complaints of the workers are shown in Table II. Gastrointestinal symptoms were both frequent and variable, but more workers complained of pain or unusual fatigue of their lower extremities than any one other discomfort (Table III).

Table I.
The Number of Employees Working with Thallium, Age at Date of Employment, Time Interval Between Employment and First Complaints, and Date of First Visit After Employment to Physician Because of Illness

Employee Number	Dates of Exposure	Age at First Employment	Time Interval Between Date of Employment and First Complaint	Date of First Visit to Physician
1	10/1/50-10/27/50 12/20/50-1/31/54*	22	3 weeks	Oct. 1950
2	10/1/51-11/13/51** 12/7/51-1/2/52	35	5 weeks	11/2/50
3	2/11/52-6/3/55	29	6-8 weeks	Apr. 1952
4	2/12/52-3/24/55	24	unknown	———
5	6/1/52-8/31/52	24	10-12 weeks	Aug. 1952
6	9/29/52-6/13/53	24	unknown	———
7	4/20/53-2/4/58*	23	4 years	———
8	6/13/55-10/31/56	27	***	———
9	6/22/55-1/17/57	20	***	———
10	11/1/56-1/17/57	24	***	———
11	4/22/57-7/22/57	30	8 weeks	7/25/57
12	4/29/57-12/18/57	22	5 weeks	6/3/57
13	9/3/57-11/27/57	41	4-5 weeks	10/7/57
14	12/14/57-1/9/58	26	4 weeks	Jan. 1957
15	12/30/57-2/14/58	24	3 weeks	2/17/58

*Thallium exposure was intermittent.
**Worked part time during May, June, August and September of 1951.
***No complaints.

Additional observations and laboratory tests were made on selected employees. The results of these are given in Table IV. As employee number 14 was in the hospital at the time of investigation, laboratory work by the Division of Industrial Hygiene was almost entirely limited to thallium determinations. Table V gives the urinary excretion rate before and after administration of BAL.

Comment

Previously reported cases of occupational thallium intoxication have occurred in European plants where the exposure has been to dusts and inorganic salts of the metal. In only one instance was it noted that the workers handled solutions as well as the dry material. This suggests that absorption may have occurred by way of the respiratory tract as well as by skin contact or incidental ingestion.

In the Ohio plant no measurable concentration of thallium in the air was found on the day of investigation. This indicates that the predominant exposure of employee number seven after January, 1957, and employees numbered 11 and 15 was chiefly through skin contact. The atmospheric concentrations in the old building where all the other employees worked had never been determined because of fire. However, the process had never changed from the beginning of operation until the time of our investigation, but a hood with an exhaust fan had been provided in the present quarters.

A solution of the same salts as used by the company studied is also employed in some analytical laboratories. In one instance it has been responsible for a case of intoxication by accidental ingestion.[12] The workers in the Ohio plant were in contact with a concentrated solution of organic salts or salts which remained on contaminated equipment. All employees had been taught to use care in handling the thallium-containing solution to prevent spillage. This was for economic rather than for health reasons.

Although symptoms appeared during all seasons of the year, there were no complaints among the workers from June, 1953, until after January, 1957, but symptoms did occur among previously and subsequently exposed men. The reason for this finding cannot be explained.

The symptoms of thallium intoxication have been widely discussed in the literature. Most authors agree that the effects are cumulative. One theory postulates that a delayed reaction may result from a single dose of soluble salt which is converted in the body to an insoluble salt and then absorbed slowly.[2] Animal experiments indicate that all salts of thallium can be absorbed through the skin. Lutz[13] found that thallium acetate acted more readily than three inorganic salts, but even the insoluble chloride proved toxic when applied to the skin in a 3% to 5% ointment. The usual symptoms in human thallotoxicosis are the same but the order of appearance will vary with the type of exposure or individual response which results in acute, subacute or chronic intoxication. The most common symptoms are nausea, vomiting, abdominal pain and cramps, pains in legs, parasthesia of lower limbs, nervousness and irritability, chest pain, gingivitis or stomatitis, anorexia and depilation which is always delayed. Except for the stomatitis and gingivitis, most of the complaints of the Ohio workers fell into this group. All other symptoms mentioned by these men have been observed by other investigators and are included in the disease syndrome.

The single differentiating symptom of thallium

TABLE II.
COMPLAINTS OF THALLIUM EXPOSED WORKERS, COMPANY X, OHIO, 1950-1958

		GI Tract								CR System		Neuromuscular System					Integument		
Employee Number	Weight loss in pounds	Anorexia	Nausea	Vomiting	Constipation or diarrhea*	Abdominal pain or cramps	Epigastric pain & soreness	Indigestion	Chest pains	Waist constriction	General Fatigue	Nervous & irritable	Backache	Pains in legs	Generalized aches & pains	Anesthesia or parasthesia	Rash	Hair loss	Remarks and Other Complaints
1	16	x	x	x	C	x	x	x	x			x		F				x	Urinary frequency; no free gastric HCl; elevated blood pressure. Had appendectomy. Disturbed sleep.
2	0		x	x	D	x				x		x	x	x	x		x		Had appendectomy. Rash appeared if he wore his work shirt a second day. Joints were swollen. Had "cold"; pallor. Faint tr. albumin in urine 8/51 and 1/3/52.
3	0	x				x	x		x			x		x[1]			x		Blood count 4/52: RBC 4.01M; WBC 6000; lymphs 29; segs 64; bands 4; eos 2; monos 2. Had had previous history of epigastric distress in 1950. GI series negative then. Rash not described but was treated 1953, 1957 and 1958 for seborrhea.
4	40										x							x	Had small area of hair loss on right temple — used "stuff" and had regrowth during employment. Skin was a "funny" color.
5	30+		x	x	C	x								x	x	x			"Difficulty thinking," depressed, "felt lost." Approved for insurance the summer of 1957. Albuminuria 12/52.
6	?+							x				x							"Difficulty comprehending"; "felt lost"; went into rages. Employment terminated following an accident at work in which he was burned with caustic.
7	0										x			x					Intermittent exposure. Rarely gets stains on hands.
11	15	x	x			x					x			x[1]		x			Stain on hands turned black and could not be removed for several days.
12	13								x		x	x		x		x			Sore throats 6/30 and 10/17/57. Released from Army 3/57. Family thought he was having trouble adjusting to civilian life. Employment terminated following repeated errors resulting from inattention to job.
13	15	x				x			x			x			x			x	Had "cough" — treated for "flu." Had hair loss in one small area above and behind one ear. Physician states he has always worried about his health.
14	25	x	x	x		x	x				x	x	x	x		x		x	Dysuria and conjestion of throat 2/16/58.
15	0									x	x		x	x	x				Off work 1/21/58-1/24/58 with "flu"; "cold" with generalized aches and pains 2/14/58.

Note: "C" denotes constipation; "D" diarrhea
"F" denotes fatigue
"?" lost weight but did not know how much
"I" especially the inside surfaces of thighs

TABLE III.
SUMMARY OF COMPLAINTS OF 15* THALLIUM EXPOSED EMPLOYEES, COMPANY X, OHIO, 1950-1958

Complaint	Number
Pains or weakness of legs	9
Weight loss	8**
Nervousness and irritability	7
Abdominal pains and cramps	7
Fatigue	6
Loss of appetite	5
Nausea	5
Vomiting	4
Paresthesia or anesthesia	4
Generalized aches and pains	4
Hair loss	4
Chest pains	4
Epigastric pains	3
Diarrhea or constipation	3
Back pains or weak	3
Constriction of chest	2
Rash	2
Indigestion	2

*No complaints made by three of the men.
**One was overweight to begin with and wanted to lose.

intoxication, hair loss, does not always occur. Most of the other complaints are not substantiated by clinical findings and diagnosis may be missed, especially if there is no history of exposure.

Since industrial thallium poisoning has been reported so sporadically in recent years, it may be well to comment on some of the findings and their counterparts in other types of exposure and theories proposed by experimental data. This may best be done by posing some questions; giving our observations and then reviewing some of the pertinent literature, especially that published since the last review by Heyroth in 1947.

1. What is the relationship between age and susceptibility to thallium?

The two chemists in Ohio who initially de-

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veloped this method of extracting diamonds from other abrasives were intermittently exposed for three to four years before anyone was employed to work on the process. Both men were over 55 years of age at first exposure and both denied experiencing any ill effects. After the process was put on a commercial scale their contact with thallium was even less although one of them periodically prepared the solution from the pure metal. No effort was made to ascertain the extent of their previous illnesses through contact with their physicians.

An age-susceptibility relationship has been known for a long time and it was recommended that children past the age of puberty not be given thallium compounds for the treatment of tinea. In the group of industrial workers studied by Meyer[6] the only man who was exposed more than three months and had no complaints was 51 years old. All the others were between 15 and 23.

There have been several reports of group exposures involving persons of varying ages. For example, in each of two families affected the oldest member survived, but deaths or permanent disability occurred at all ages.[14,15]

In one of these families a 38 year old woman who was five months pregnant, died.[14] On autopsy thallium was found in her spinal cord, liver, kidney, vagina, uterus and bladder. None was found in the kidney or liver of the fetus.

Three other cases of thallium intoxication in pregnant women have been reported. One woman, who had used a dipilatory cream, conceived after onset of illness, and a therapeutic abortion was done.[16] A second woman became ill January fourth when she was four months pregnant.[17] A fetal heart beat of normal rate was first heard in the middle of March, even though the mother had tachycardia for another two months. An apparently normal baby was delivered to a still paralyzed mother, on June tenth. This author was unable to answer his own questions as to whether the baby was unaffected because of the apparent age-susceptibility relationship or if the placental barrier protected the child. At time of delivery no thallium was found in maternal or fetal urine, placenta, membranes or amnionic fluid.

There was an earlier report of a woman who ingested 1.2 gm of thallium at term.[18] The mother was hospitalized 89 days with colic, diarrhea and polyneuritis but the baby died five days after birth. Thallium was found in the baby's tissues.

German authors, as recorded by Sollman[2] have stated that thallium passes to the fetus and that milk of poisoned animals may be toxic. More recently[19] thallium was found in urine and meconium of infants whose mothers had absorbed thallium during the seventh month.

2. Does the blood picture aid in the diagnosis of thallium intoxication?

Eight of the men in the Ohio study had had blood counts during their illness or immediately following their exposure. The white counts ranged from 5,300 to 10,900. Except for employee number two, whose differential ranged from 0-7 eosinophiles and 20-25 lymphocytes during his second hospitalization (total eosinophile count was subsequently found to be within normal limits), the differentials were 1-4 eosinophiles and 29-42 lymphocytes. There was little change in total or differential counts done on five of the men seven months to four years after last exposure.

TABLE IV.
BLOOD PRESSURE AND LABORATORY FINDINGS ON SELECTED THALLIUM EXPOSED WORKERS, COMPANY X, OHIO

Employee Number	Dates of Observations	Blood Pressure	Blood Count							Mg. Thallium per Liter of Urine	Remarks*
			Hb	RBC (Millions)	WBC	Differential					
						Lymphs	Neutr	Bands	Eos	Misc.	
1	2/13/58**		15.5	4.88	8400	36	58	2	4		none	
	3/13/58	180/110										Urinary lead 0.025 mg/liter
7	1/24/58									Basos	0.164	
	2/10/58		14.0	4.38	7900	32	65	2		1		
	3/1/58	130/80									none	
11	1/24/58									Monos	0.090	
	2/10/58		14.0	4.35	6100	34	52	6	4	4		
	3/2/58	130/70									0.025	
	4/13/58										0.054	
12	2/13/58		17.0	5.29	10000	14	77	4	2	Basos 3	none	
	3/1/58	132/70										
13	11/20/57***		14.4	5.00	10400	33	64		2			
	2/24/58	130/80									none	
15	1/24/58										0.921	
	2/15/58		15.0	4.19	5400	42	52	2	4		0.530	
	2/24/58										none	Tr. albumin in urine****
	3/2/58	130/80									none	

*All smears were negative for stipple cells.
**See history, for additional laboratory and blood pressure readings.
***From his physician's record.
****All other urine specimens were negative. All were negative for sugar, hematin, bilirubin and porphyrin.

Many investigators consider that a leukocytosis with eosinophilia and lymphocytosis has been accepted as typical,[1,3,7,19] however, von Martius[20] reported no typical blood picture but a tendency to an increased white count and a lymphopenia.

3. Does thallium intoxication cause an elevation of blood pressure; and if so, how permanent is the effect?

Only three men in the Ohio study had records of blood pressure readings taken during their acute illness. Hospital admission recording for employee number two was 120/70 for first admission and 160/80 at second admission; for employee number 14, the blood pressure was 160/90. There were no additional records during the hospital stay for either patient.

Employee number one had had no illness since childhood, and his only physical examinations were those done in the Army. While overseas, he was told that his blood pressure was elevated but after a few minutes rest, the reading was satisfactory. His mother and two of her three sisters have been treated for hypertension. He was admitted to the hospital four weeks after his first exposure to thallium. During his two-week hospitalization his blood pressure ranged from 150/108 to 180/120. On November 17, 1950, one week after discharge, it was 120/70. He returned to work about the first of December and in January, 1951, his blood pressure was 130/80. He worked with thallium daily, but not always all day, until January, 1954. In May, 1953, his blood pressure was 170/90, in December, 1954, 170/110, and in March, 1958, several readings ranged from 150/90 to 180/110.

Blood pressure elevations have been reported in patients with acute and subacute thallium intoxication.[15,21,22,23,24] Most of the cases reported resulted from ingestion of rat poison, although the source of exposure was unknown in five. Of Allsop's 18 cases, one moribund patient had a subnormal blood pressure while 13 had diastolic pressures of 90 mm. Hg. or above and one patient left the hospital with a reading of 160/100. Winter's nine year old patient was found, on the eleventh day of hospitalization, to have a blood pressure of 170/145. For the next month it ranged from 140/100 to 180/165, the systolic going even higher following a convulsion. Four of the five young men observed by Mertens had blood pressures of between 160-200 mm. Hg. systolic and 110-140 mm. Hg. diastolic following a single dose of a thallium salt. The blood pressures returned to normal in Winter's and Merten's cases in about two months. None of the cases previously reported had known multiple exposures to thallium.

4. What is the relationship between albuminuria and exposure to thallium or the appearance of symptoms?

Four Ohio workers were found to have an albuminuria during their exposure or illness. Employee number two had a faint trace of albumin in his urine two months following his first exposure which was on a part-time basis. One month after starting to work full-time, the urine was negative but was again positive during his second hospitalization.

Employee number five's physician wrote that he remembered that during the patient's acute illness, there was both albuminuria and hematuria. The first entry on the patient's medical record was two months later, at which time the urine was negative. In still another two months, the patient failed to pass a pre-employment physical because of albuminuria.

Employee number 14 had a trace of albumin the day after hospital admission, January 16, 1958. On the twentieth it was negative but on February 10, 1958, was found to be 3 plus; on the thirteenth it was one plus and was negative for the next three consecutive days.

A trace of albumin was found in the urine of employee number 15, eight days after last exposure but was negative on the last day of exposure and one occasion before that.

Winter[24] also reported finding albumin in the urine of his patient on only four days, although the urine was tested almost daily. This would suggest that albuminuria may occur intermittently with thallium intoxication. Carrozi had reported that albuminuria appears following exposure and disappears after termination of work.[3]

5. Is there an impairment of liver function with thallium intoxication?

Only one man in our series (employee number 14) had liver function tests performed. The thymal-turbidity test four days following admission was within normal limits but cephalin flocculation was positive. This latter test had returned to normal three weeks later, although at that time serum transaminase was slightly elevated.

Van Oettingen[19] reports that liver impairment is rare. Although yellow atrophy and fatty degeneration has been reported in the literature,[11,15] reports of liver function tests in recent literature is scanty.

6. To what extent is eye damage related to thallium intoxication?

Although there was no optic nerve damage evident when examinations of two Ohio men were done, one had an unsustained nystagmus.

As mentioned before, optic atrophy has occurred as a result of both industrial exposure to thallium and from skin absorption of thallium-containing dipilatories.[1,4] Other authors have

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reported eye damage following unknown exposures, or intoxication following ingestion.[23,25,26] Central scotoma, keratitis and cataracts have also been reported.[7]

7. How can mental symptoms be related to exposure?

Several of the workers in the company investigated complained of irritability but none sought medical aid for this symptom. This complaint was verified by their co-workers or families, who stated the men, who had formerly been jovial and easy-going had become very touchy and difficult. Three of the men complained that they "felt lost" and had difficulty comprehending.

Mental symptoms have been reported to accompany known cases of thallium intoxication.[15,27,28] The first three patients in Neal's[14] series were thought to have had encephalitis following varicella until other members of the family became ill. Schutzler[25] described four cases of thallium intoxication which were given psychiatric diagnoses because the physical symptoms were bizarre and objective findings were negative and psychiatric symptoms were evident. He later[28] reported two cases of thallium poisoning with predominantly mental symptoms with source of exposure undetermined. The damage in these two patients was permanent, as evidenced by electroencephalograms and pneumoencephalograms. In another series electroencephalograms of five of seven patients tested were abnormal.[23]

8. How frequently is rhinitis, conjunctivitis or sore throat the presenting symptom of thallium intoxication?

Only one of the workers in the Ohio plant visited his physician mainly because of a sore throat. He had had pains in his legs which were relieved temporarily after being treated with penicillin. Another was diagnosed as having "flu"; the onset of his symptoms coincided with an influenza epidemic in the city. A third felt (and looked, to this observer) as if he had a cold.

Fairweather, *et al*[29] stated that early signs of rhinorrhea and conjunctivitis are often ignored. Grulee and Clark[30] reported these symptoms in three of their four patients. An original diagnosis of tonsillitis was made in the cases presented by Welty and Berry.[31] The victim in these last three mentioned papers were children, some of whom were known to have eaten poisoned bait; the kind of exposure in the other cases was not given.

9. Is achlorhydria frequently associated with thallium intoxication?

No free hydrochloric acid was found in the one worker (employee number one) on whom a gastric analysis was reported.

Although epigastric pain is a frequent complaint of persons with thallium poisoning, and achlorhydria is an accepted sign,[19] there are very few recent observations reported on gastric analyses. Von Martius[20] reported on 13 people who had ingested thallium. All had hypoacidity; followed in one by hyperacidity.

10. Would observations of serum calcium levels be helpful in making a diagnosis?

The only patient in the Ohio study on whom this observation was made had a definite elevation of serum calcium on first analysis. A repeat sample contained slightly more than the value given for the upper limits of normal.

Elevations of serum calcium are given as evidences of thallatoxicosis,[2,19] but instances are not cited in any of the more recent literature.

11. How effective is BAL in the treatment of thallium intoxication, and how does it affect urinary excretion?

Two of the patients in the Ohio series received treatment with BAL. Employee number two had no noticeable relief of symptoms after five days of administration. No excretion data is available on this patient. Employee number 14 complained of return of abdominal pain, nausea and vomiting after initiation of treatment. Unfortunately, the urine sample just prior to treatment was lost, but by extrapolation it does not appear that the drug caused any great increase in the urinary excretion of thallium (Table V).

BAL was used experimentally as early as 1946 and found to be ineffective in increasing the tolerance of animals to thallium. The experiments of Moeschlin and Demiral[33] indicate that the giving of BAL is probably useless and Mazzei and Schaposnik[21] felt that this treatment caused increase in the pains in the limbs of their patient. On the other hand, Diengott[34] reported improvement in three days after beginning BAL treatment of a patient a month after ingestion of 30-40 mg of thallium. A 50% increase of the excretion of thallium acetate during the first eight days following ingestion has been reported to be effected by the administration of BAL.[35] Several German investigators (reviews in *Chemical Abstracts* and *World Medicine* and not all authors noted), have found BAL effective only if given very shortly after intake of the metal, and Lund[36] in 1956, following experiments with different chemicals recommended that BAL not be used in the treatment of thallium intoxication.

Detailed data on the excretion and distribution of thallium is not available; however, some work is being done with radioactive thallium.[37] Thallium has been detected as long as three or five months[38] following ingestion and several authors have reported serial determinations on urinary concentrations.[8,17,23,39]

A frequent complaint encountered by many investigators is stomatitis and gingivitis. Not one of our group could remember having a sore mouth at the time of their illness or exposure.

Tachycardia, another frequently reported sign, was not specifically noted in any of the Ohio workers.

Despite the fact that nervous symptoms are pronounced, there are few reports on abnormalities in the cerebral spinal fluid, and these reports[24,25] do not give the chemistry of the fluid.

TABLE V.
URINARY EXCRETION OF THALLIUM — EMPLOYEE NO. 14, COMPANY X, OHIO, 1958

Date	Mg. Tl/liter	Mg. Tl/24 hr.
1/23-24/58	1.000	1.700
1/24-25	0.977	0.760
1/25-26	1.871	1.460
BAL started 1/30		
1/30-31	0.470	0.647
2/1-2	0.766	0.804
2/2-3	0.380	0.723
2/3-4	0.356	1.030
2/4-5	0.550	0.578
2/5-6	0.700	0.730
2/6-7	0.540	0.620
2/7-8	0.190	output unknown
2/8-9	0.240	0.264
BAL discontinued 2/11		
2/11-12	0.196	0.301
2/15-16	0.320	0.253
2/16-17	0.132	0.092
2/21	0.126	0.095 (18 hr.)
2/21-22	0.204	0.155 (8 hr.) overnight
2/22-23	0.170	0.136 (29 hr.)
2/24-25	none found	
2/28-3/1	none found	
3/8-9	none found	

One of the men in our study had elevation of protein and sugar in his cerebraspinal fluid.

There have been other signs and symptoms reported by different authors, and Mertens[40] stressed the multiplicity of signs and symptoms which might appear. Among these are disturbances of the nervous system and ductless glands. No observations on malfunction of endocrine glands were made by any of the Ohio physicians of the men in the present study.

Prevention

Since all compounds of the metal are very toxic by any mode of entry into the body, the greatest care must be exercised to prevent exposure.

The following recommendations are made to aid in preventing the occurrence of occupational illness. Essentially they differ only slightly from those made by Carozzi[3] and later by Sessions and Goren.[41]

1. *Worker education:* This should include information on the dangers encountered in the use of thallium to promote safety consciousness and the reporting of illnesses or accidental exposures so medical evaluation can be made; job procedure and use of equipment and housekeeping practices to minimize continuous or accidental exposures; and personal protection by exercising healthful personal habits and the proper use of protective clothing. Needless to say, a good educational program can only be attained if management is aware of the toxicity of the products purchased for use in industrial operations.

2. *Engineering controls:* Where the necessity for control of a hazardous process is foreseen in the planning stage, engineering controls such as process change, substitution of materials, isolation of hazardous processes, and design of buildings and equipment for minimum toxicant generation and air contamination may be incorporated in the original design of the installation.

In the usual instance, however, the controls must be tailored to processes and equipment already installed and operating. Consideration must then be given to the requirements and limitations imposed by the raw materials and manufacturing methods and by the dictates of process and production economics. This usually limits engineering control to a combination of several of the following methods; segregation and/or enclosure of process, alteration of equipment, building and equipment maintenance and local and general exhaust ventilation.

3. *Medical controls:* All persons should be under medical supervision. No one should be employed to work with thallium if he has ever had a history of having albuminuria, high blood pressure, hepatic disease, endocrine gland disfunction, blood dyscrasias or mental instability. Periodic physical examinations should include interval history, urinalysis, complete blood count, blood pressure, eye examination and observation of body weight. Interval between examinations must be determined on an individual company basis depending on the amount of exposure or on an individual employee if he has developed symptoms. It is important that there be a close working relationship between plant and personal physician.

4. *Protective clothing and equipment:* Special clothing, including rubber gloves, should be provided to prevent skin contact. Clothing should be kept in a locker separate from street clothes and washed frequently. Gloves should be washed and masks cleaned with each wearing. Contaminated clothing should not be taken home, but washed in company or commercial laundry with the minimum of handling.

5. *Washing facilities:* Adequate washing and shower facilities should be provided and their use made mandatory.

6. *Personal habits and hygiene:* There should be no eating, loafing, smoking, or gum chewing in exposure areas. Hands should be washed be-

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fore handling food or cigarettes and showers taken at the end of the work day.

7. *Housekeeping:* All working surfaces should be kept clean. Copious amounts of water should be used following accidental spillage of thallium compounds.

Summary

A study was done of a plant where 15 men had worked with varying concentrations of solutions of organic thallium salts in a 7½ year period. All but three had complaints which may have resulted from their exposure and diagnosis of thallium intoxication was eventually made in six.

The chief complaints were abdominal pain, fatigue, weight loss, pain in the legs and nervous irritability. Three of the men were originally diagnosed as having appendicitis and surgery was done on two of them. Albuminuria occurred in three patients, and hematuria in one; abnormal liver function test in one and elevated cerebral spinal glucose and protein in one. The blood pressure of a 22 year old man was elevated during his hospitalization for an appendectomy but returned to normal in a month. He returned to work with thallium for another three years; during the last eight months his blood pressure remained elevated and after four years in another occupation was still abnormally high. He had, however, a history of having had on a single occasion a slightly elevated blood pressure prior to first exposure to thallium.

Thallium intoxication can present diagnostic difficulties, especially if the history does not reveal any contact with the metal. This is true in early cases of chronic poisoning because subjective symptoms are not necessarily accompanied by clinical signs or positive laboratory findings.

There is not adequate information on certain laboratory tests to ascertain which would be the most useful aids in establishing a differential diagnosis.

Urinary excretion of thallium is rare in unexposed persons but no relationship between excretion rate and either exposure or the appearance of symptoms has been established.

BAL did not effect prompt relief of symptoms in the two Ohioans treated and apparently did not materially alter the excretion rate in the one on whom data is available.

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Byssinosis—History and Investigation

For nearly 200 years Lancashire, Great Britain, has been identified with cotton manufactures. While these brought wealth and industry to the population of the crowded mill towns, they also caused an inordinately high morbidity and mortality from chronic respiratory disease. The whole community suffered excessively from bronchitis, which was nonoccupational but due to pollution of the atmosphere by the acrid smoke of house and factory chimneys. Certain groups of workers, notably those employed in in the processes of preparing the cotton and in the cardrooms and blow-rooms suffered additionally from "asthma," which was occupational and popularly attributed to the irritation of cotton dust. J. Leach, a certifying surgeon of Heywood, Lancashire, described the disorder in 1863. Since then the disease, byssinosis, has continued to occur, causing chronic ill-health, disablement and death. In recent years byssinosis has been the subject of extensive researches — clinical, pathological and epidemiological — by R. F. S. Schilling and his co-workers, who have been admirably assisted by the mill owners and by the trade unions. Schilling and his colleagues concluded that the symptom complex of chronic bronchitis is not the same as that of byssinosis, for whereas men with chronic bronchitis develop tightness of the chest whenever they are exposed to an irritating concentration of any dust, in the early stages of byssinosis the workers are affected only by cotton dust and only on Mondays or on the day immediately following absence from work. They divided the disease into two stages — grade I in which chest tightness and/or breathlessness occur on Mondays only, and grade II in which chest tightness and/or breathlessness occur on Mondays and other days. This means that the occurrence of Monday symptoms previously called "Monday feeling," is the specific characteristic in the diagnosis of byssinosis. In 1956 the Industrial Injuries Advisory Council reported that although an experienced observer visiting the cotton mill on a Monday might be able to decide with some assurance whether a person was experiencing the symptoms of byssinosis (grade I), there was no simple objective test by which the disorder could be distinguished. C. B. McKerrow and his colleagues have recently sought to discover such an objective test. In a carefully planned study they measured respiratory functions by the accepted modern methods; these included indirect maximum breathing capacity (M.B.C.), maximum voluntary ventilation (M.V.V.), and peak inspiratory and expiratory flows coupled with an airways resistance (A.W.R). The results showed distinctive specific patterns for both grades of byssinosis and for the controls. McKerrow and his colleagues concluded that the mill dust — quantitatively as well as qualitatively — is the cause of the respiratory impairment and that it contains some pharmacologically active constituent. This work marks an important advance towards the establishment of a practical objective test for the detection of the earliest stage of the disease.

—From *British Medical Journal*, July 26, 1958.

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The initial appeal of early and new shot towers springs from the traditions of metallic poisonings — lead, arsenic and antimony. Actually, few or no metallic poisonings are recorded. Those mentioned nebulously chiefly are to be identified with the dross from the heating of the metal prior to pouring. The true appeal of old shot towers stems from their dramatic place in the history of industrial America. For the moment, waving aside all the potentialities of occupational diseases, this is the story of shot towers themselves.

Lead Shot Towers in America

OLD AND NEW

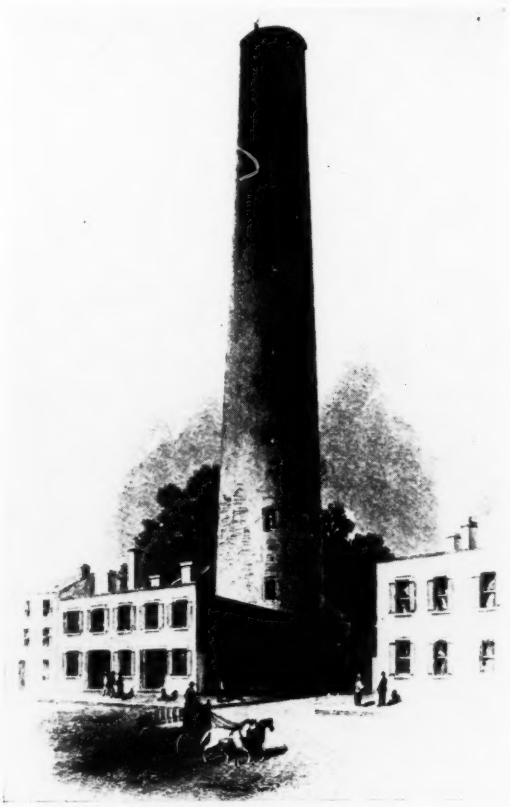
CAREY P. McCORD, M.D.

Of all industries that have survived 200 years or more, shot pouring has changed least. The first shot poured and the last relied upon strikingly similar procedures. The oncoming decades have left untouched the basic principles, adding refinements only in power, sources of heat and mechanical handling. The first professional shot pourer of 1769 in Bristol, England, might walk into the most modern of shot towers and take over after learning to press a few electrical buttons. In truth, the first of all man-made shot towers, still standing and still operating, has undergone so few alterations as to attract no attention in the neighborhood.

Devotees to the shotgun may be reckoned by myriads. Their tenderness for shotguns is akin to that of Abélard for Héloïse. For most of them, shot are but lowly pellets fired almost as excreta from the artistry of their affection. Such scorn is little deserved. Shot made the shotgun. The antecedents of the modern shotgun were scarcely prideful objects. The blunderbuss, the musquetoon and the harquebus earned their reproachful designation "slug guns." Later came the precision and exactness of poured shot, and finally came the the fineness of shotguns that spurred to rapture. As much romance attends shot making as is now larded upon the gun that fires those historic missiles. Here is the evidence.

The skyline of more than one early American city was dominated not by lofty church spires but by shot towers. This was true of Philadelphia, of Baltimore and of New York. In time the total exceeded 25. Never was it necessary for the visitor to inquire "Where is the shot tower?" Always it was in sight — the tallest structure in town, and sometimes the most beautiful.

In Philadelphia, Bishop, the good Quaker, was proud of the shot tower that he and his plumbing partner, Sparks, erected in 1808. Well he might have been proud. That was the first substantial one in the country, and the partners had bested



Bishop-Sparks Tower, Philadelphia.

From the Institute of Industrial Health, University of Michigan, Ann Arbor, Michigan.

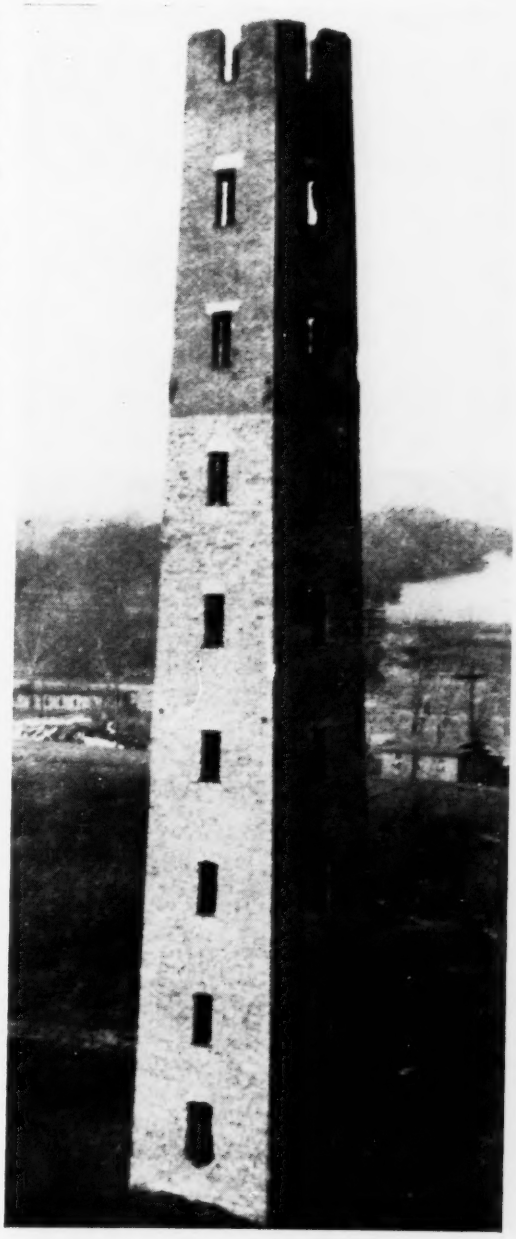
their competitor, Paul Beck, as to the time of the first shot pouring, by only one week. The Bishop-Sparks tower was completed on October 20, 1808, while that of Beck was finished on October 27. Yet, Beck had his own victory. His square stone tower at Twenty-First and Cherry Streets on the Schuylkill River was taller by 24 feet. The Bishop-Sparks round brick structure on Carpenter was only 142 feet in elevation, but it extended 30 feet below the ground level. What Bishop and Sparks could not have known was that by 1823 the Beck tower no longer would be in operation while their own was destined for nearly a century of shot making; that long after its shot making days were over, and to this day well intact, it would remain one of Philadelphia's proud landmarks; and that the majority of the near score of other shot towers shortly to follow the Philadelphia enterprise soon would cease shot manufacture.

Above all, that which might have pleased most, could they have known, was that nearly 150 years later, in a land of extraordinary industrial ingenuity, all dropped shot still would be made by the same fundamental methods used by the Philadelphia groups in 1808. Only mechanical refinements have appeared. Needed only, then or now, are high elevation, molten lead with added metallic arsenic to promote fluidity, a colander and a vat of water at the bottom for quenching. Nature's physics are the real workers.

All did not remain well. Soon came the War of 1812. Bishop, the Quaker, was a pacifist. No ware of his was to be any implement of warfare. He felt he must retire, and did. Shotguns had no real future as military weapons, but there was a minor military past, such as with King Edward's 300 Flemmings, fighting in 1471, and at the siege of Parma in 1571. Sparks, the partner, without hampering religious scruples, quickly turned to military missiles of molded bullets and balls and to the undroppable buckshot. Perhaps that is what the devout Bishop feared all along.

So the Bishop-Sparks tower became the Sparks tower, while the Beck tower, soon to become an industrial casualty, became better known as the Schuylkill tower. That tower was not to be without its day of glory. On February 15, 1815, two days after the announcement of the Ghent peace treaty, Philadelphians joyously celebrated around their tallest structure, Beck's tower, which was gaily decorated by mayoral proclamation with 160 brilliant lamps. Afterward both towers reverted to more emphasis on shot for the "birding" and the "fowling" pieces. Left alone, these two towers, with a daily capacity of some 10,000 pounds of shot and a revenue of 200,000 dollars annually, could have met the country's requirements for some decades. But they were not to be left alone in any such monopoly.

That word "patent," in an earlier paragraph, is significant. In that period anything that was "patented" was accepted as something unique, mighty or magical. The Philadelphia shot manufacturers stressed the word "patent" or "English patent" in their advertising. That relates to the William (or Josiah) Watts' patent which by that time had expired, but the word remained potent. The date of Watts' patent, which ran for 14 years, usually is stated to be 1782. This patent was known in the United States at least as early



Shot Tower, Dubuque, Iowa.

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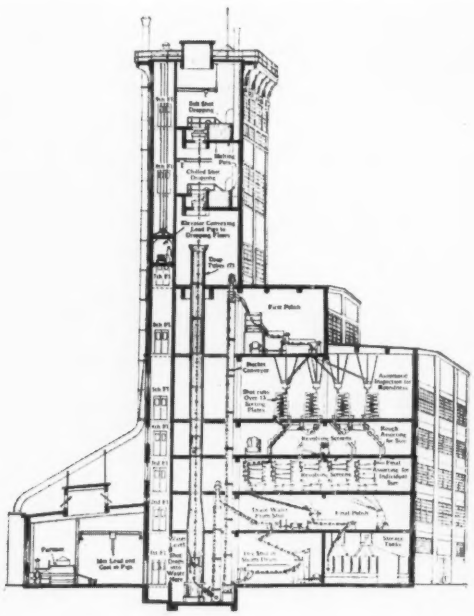
Paul Beck's Shot Tower, near Schuylkill.

Bishop-Sparks Tower, Philadelphia.

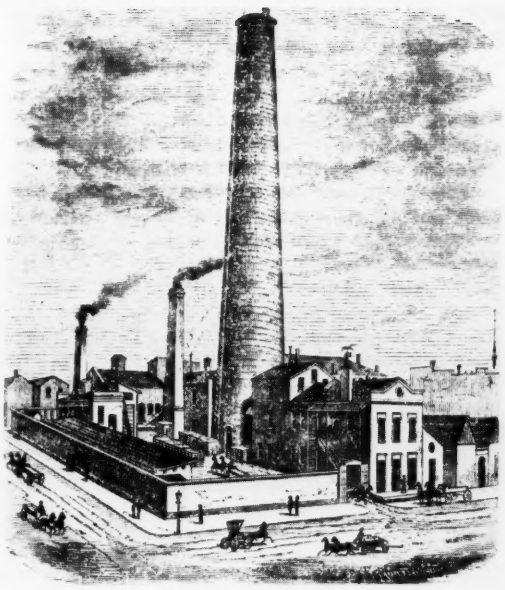
as 1789, as indicated in the Moses Austin papers, following record examination in London by Henry Miller.

Moses Austin, of Connecticut, sometimes identified as "a pioneer of Virginia," was a merchant in Philadelphia, then a pewter maker in Richmond. Later, beginning in 1789, he carried out extensive lead operations near Wytheville and Austinville, Virginia. There he made shot, as shown by his invoices of 1794. The petty volume suggests that the shot were not dropped. The substantial shot tower now standing at Jackson's Ferry was not erected until 1820 — long after Austin's departure. In 1796, Austin first entered Missouri, and by 1799 he is credited by Schoolcraft with having a shot tower in operation near Potosi, under the superintendence of his nephew, Elias Bates. This being true, Moses Austin was the first commercial dropped shot manufacturer in the country, antedating the Bishop-Sparks enterprise by some nine years.

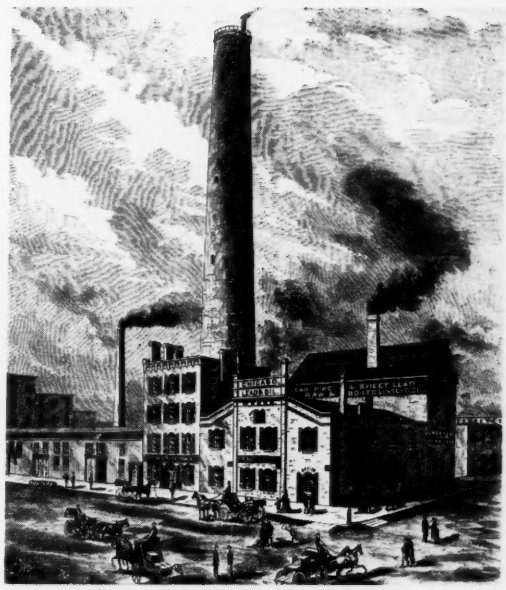


Exterior, Olin Mathieson Chemical Corp. Shot Tower, East Alton, Ill.

Diagram, Modern Winchester Shot Tower, Olin Mathieson Chemical Corp., New Haven, Conn.



St. Louis Shot Tower.



Chicago Tower, Clinton and Fulton Streets.

Vehement protests are prone to arise whenever William Watts is mentioned as the "discoverer" of shot dropping. Dissenters contend that for generations before Watts, shot pouring was a common skill. The origin trails off in mistiness toward the Orient. Before Watts' time, shot customarily were made through two laborious procedures. In the first of these, sheet lead was cut by hand into tiny cubes and placed in a tumbling machine, a "rumbler," along with some abrasive, or perhaps with nothing more than plumbago for polishing. In good time these dice reached some degree of roundness and became "shot." In a second method, shot were cast, as are buckshot, in a multilocular mold. Fifty to 70 mold spaces were known but were not common. The whole process was something like the pouring of molten lead into the cells of a hornet's nest. At best, every shot ended up with a sprue, or stem, to be trimmed. Perhaps more slugs than shot were fired. The term "slug gun" may not have been defamatory.

Acclaim does not always become the boon of the discoverer. That may await the later person who through some procedure captures the imagination of a public ready to praise. Discoverer or no, Watts did just that. When, in 1769, he stood on a tower of Saint Mary Redcliffe church in Bristol, England, and successfully dropped shot, a world was ready to shout. Out of that experiment came a patent, the first substantial shot tower, fame for Watts and what to him seemed a fortune in money, which promptly slipped through his fingers. That day at Saint Mary Redcliffe paved the way for an industry — an industry still prospering. Watts' detractors, then and now, grudgingly do make two concessions. He may have been the first to drop shot from a great height, for the making of bigger shot size, and beyond debate he was granted a patent.

In 1956, questing far and wide for shot tower lore, I journeyed to Bristol and Saint Mary Redcliffe. I wanted to determine the exact spire and the exact level from which Watts poured his first shot. While Saint Mary Redcliffe has published its history in a handsome volume, not a word was mentioned of Watts and dropped shot. Those consulted were amazed at my representations. Yet directly across the street from Saint Mary, not 100 feet away, stands and still operates the first of all shot towers. What was inaugurated on that day in 1769 at Saint Mary's has touched many more hundreds of thousands of lives than any other single event staged at that historic edifice.

Prosperity from shot dropping in Philadelphia and Missouri prompted the creation of many other shot towers. All too many, since in time the number led to the undoing of all the old ones. Baltimore provided four shot towers. Herculaneum, Missouri, and New York City each provided three. Towers appeared at Jackson's Ferry near to Wytheville, Virginia; at Lexington, Kentucky; Helena, Wisconsin; Dubuque, Iowa; St. Louis, Missouri; and Chicago, Illinois. Extravagant claims were made about nearly all these towers. Indeed all were "firsts" somewhere — the "first" beyond the Alleghenies, the "first" beyond the Mississippi River, etc. Several claim

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to be the tallest when, in truth, no other has equaled the 349 foot height of the Villach, Carinthia, tower in Austria. The Chicago tower may have been the first to install an elevator, modern for its time. These towers, in little known and then sparsely settled sections, were not necessarily operated in petty volume. The Helena plant during the years 1855-1859 turned out more than 1,400,878 pounds of shot.

In the early days, shot were dropped not only from especially constructed towers, but down mine shafts, in quarries and even from the Natural Bridge in Virginia — anywhere the required height could be found. As late as November, 1814, William Caruthers, of Lexington, Virginia, visited Thomas Jefferson at Monticello and secured the right to use the Natural Bridge for making shot. This bridge, 14 miles from Lexington, spans the Cedar Creek. Its height is 215 feet, and thus is ideal for shot pouring.

The towers near Herculaneum and in Baltimore, while not representative and utterly unlike in structure, warrant more detail. The Herculaneum towers actually were not man-made towers at all. Platforms were projected over the top of the tall bluffs of the Mississippi River, the height being about 170 feet. One of the three towers there was the second of Moses Austin. His first was already located only 35 miles away at Mine-a-Burton near Potosi. One workman on any one day could drop 4,000 to 5,000 pounds of shot, but polishing was another matter — one day of dropping and nine days of finishing. In the War of 1812, the towers around Herculaneum well served the armies in the Gulf country, not so much by dropped shot as by bullets, buckshot, and other lead materiel. The importance of the towers to the economy is best revealed in the fact that one tower was portrayed on the local greenback money.

Of all the surviving old shot towers of the country, the Merchants Tower in Baltimore, at Fayette and Front Streets, is the most imposing. Not the oldest in the city, it followed by five years the Wolfe tower on North Gay Street. This Merchants Tower, also known as the Phoenix Tower, was erected in 1828. It was, and is, the centerpiece and heart-piece of the city. This brick structure stands 234 feet above the street level. Its diameter at the base is 40 feet, at the top, 20. At the base its walls are four and a half feet thick. The cornerstone was laid June 2, 1828, by Charles Carroll, the last survivor of the signers of the Declaration of Independence. By November twenty-fifth of that year the tower was completed — 1,100,000 bricks had been laid leading to 14 floors, counting the parapet.

Baltimore's third tower, on Eutaw Street, was demolished in 1851 after an earlier fire. The Wolfe tower was torn down about 1845. The fourth tower on Howard and Montgomery Streets was not erected until 1877.

Compared to the massive and graceful Merchants Tower, the other three were pygmies, not alone as to stature, but as to city affection. Here the output capacity was 500,000 bags per year. But how heavy was a bag? Four months after the Phoenix (Merchants) Tower was completed, Edgar Allen Poe was in Baltimore. Apparently he was bowled over by the impressiveness of the tower. On March 31, 1839, he caused to be printed in the daily press a notice that on the following day a man would fly from the top of the tower to Lazarette Light House in the Bay. A huge crowd assembled for this miracle. Slowly came the realization that it was All Fools' Day. Long after this tower's work days were over, through popular subscription of 14,500 dollars, it was purchased as a civic monument.

Simple as are the physical requirements for shot pouring, the commercial production of uniform shot was far from trouble-free. In the early towers the molten lead, with its low arsenic content (about 1%), was prepared at the base of the tower in an adjoining space, otherwise the tower would have become a smokestack. Some did. The old tower at Lambeth, England, was notorious for its perilous wall incrustation of arsenic, lead and sulfur. The fuel was wood or charcoal. From the beginning all substantial operations utilized some form of hoist, if not more than a windlass. The size of the perforations in the colander determined the size of the shot, but at first not accurately. The cleaner the sieve, the less uniform the shot. This was overcome by a bed of loose lead oxide so that the molten lead seeped rather than streamed. The upper limit of shot size, conforming to the laws of physics, was about 0.23 of an inch. These largest sized dropped shot sometimes were called "buckshot" — that not being a fixed term. At least eight sizes of buckshot are recognized, but the true buckshot required molding or swedging. The smallest shot size was 0.04 of an inch. Such shot could be poured from platforms far below the top of the tall towers. Some shot pouring took place at various tower levels, the largest shot being dropped the longest distance. After pouring, sorting and grading became a task.

The method of sorting shot was as ingenious in its utilization of physics as shot dropping itself. All that is required is a series of large, sloping, staggered plates arranged with spaces between. Well-rounded shot will roll in a straight line, acquire momentum, leap over spaces between successively lower plates, and end up in a receptacles as perfect shot. Imperfect ones roll to one side and drop off the lower plate rim to be remelted. The slugs don't roll.

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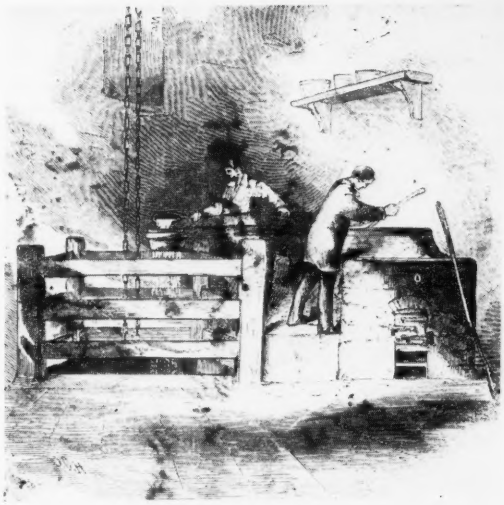
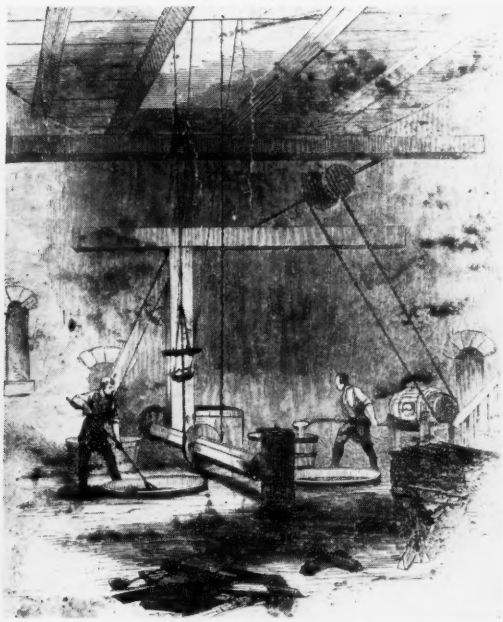
St. Mary Redcliffe Church, Bristol, England, Where the First Shot Were Poured in 1769.

Through this Dropping Pan Flows Molten Metal at the Winchester Plant of Olin Mathieson Chemical Corporation, Pellets Taking Spherical Form and Dropping 154 Feet into a Tank of Water.



Furnace Room of Merchant's Tower, Baltimore, Showing Lead Melting and Pouring.

Dropping Pan for Number 6 Shot at Winchester Plant of Olin Mathieson Chemical Corporation.



Melting Tubs in the Merchant's (Phoenix) Shot Tower, Baltimore.

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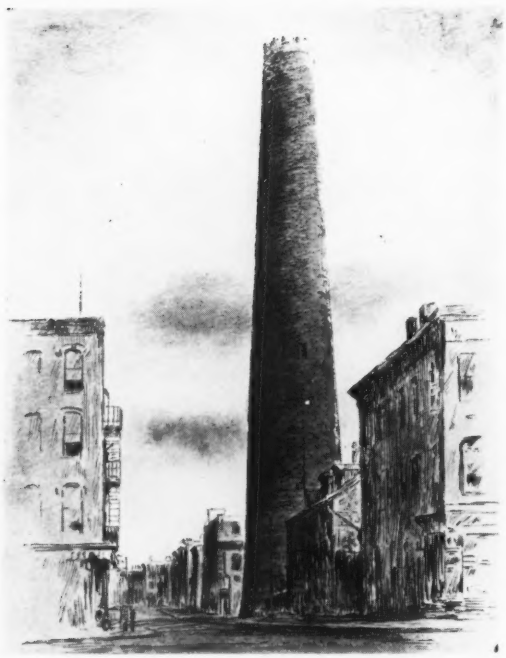

Merchant's (Phoenix) Shot Tower, Baltimore.

All lead shot quickly tarnish owing to oxidation. This leads to polishing and lubrication. Many modern shot are copper plated to escape the unsightliness of tarnish. Contrary to earlier difficulties, modern shot pouring, a continuous operation, is conducted with such calculated precision that uniformity is the rule, slugs and imperfections the exception. The production rate may be more than 10,000 shot per second.

The printed "obituaries" of shot towers are prone to state: "The methods in use proved to be obsolete." Pure apology. The method remains the same, only the procedure changed. Today more shot than ever are poured. Every year more than 40,000 tons of lead end up in those tiny pellets cherished by the shotgun sportsman. Some riflle worshipers, with disdain for shotguns and shotgun shooters, created the contemptuous "shotgun sniveller." But those addicts, undismayed, go their own happy way, yearly firing billions of dropped shot. Why then the passing of all the old shot towers into historic monuments, and why the literal downfall of many? There is no mystery. In earlier decades every region of the country was nearly self-sufficient in meeting its needs. Just as the slaughterhouse and the sawmill were essential on a community basis, so on a regional basis was the need for a shot tower. For the country store, the traditional symbol long was the cracker barrel, but the shot bag was just as appropriate a symbol. Shot were sold everywhere, and in at least two sizes, "bird" and "squirrel." Better transportation doomed the regional shot tower. More important than that, with breach-loading shotguns the time came when the hunter did not want shot alone. He wanted gun shells complete with powder, primer, wads — everything. Right there was the nemesis of the old shot towers. The ammunition makers' day had arrived.

The output of the five modern shot towers in the United States, (New Haven and Bridgeport, Connecticut; East Alton, Illinois; Kings Mill, Ohio; and San Francisco, California) now operated by ammunition manufacturers with their marvels of mechanization, can outdo in costs, volume and quality 100 old-time towers depending on rock bluffs, abandoned mine shafts, hand-wound windlasses, and wood-fired melting pots. Some of the industrial romance has passed, but not the fundamentals. The modern tower may turn out dropped shot in endless fecundity, but the four essentials of shot making remain — molten lead, a sieve, height, and a vat of water at the bottom.

Industries Covered by Industrial Committees

ILO Industrial Committees have been set up for the building and civil engineering, chemical, coal mining, inland transport, iron and steel, petroleum and textile industries, and the metal trades, and all of them have called for investigations into the safety and health problems of their industry. Among the subjects on which the Office has recently prepared studies for the Committees are shift work in the chemical industries, health risks in the building industry, safety and hygiene in the chemical industries, the classification and labelling of dangerous substances, welfare facilities for dock workers, safety in coal mines, occupational diseases in the chemical industries and hygiene in the textile industries. For some industries the Governing Body instead of setting up permanent industrial committees may convene ad hoc tripartite meetings. A tripartite meeting was held in 1957 to discuss various labor questions in mines other than coal mines. The questions included accident prevention. A tripartite meeting will probably be held in 1958 to discuss labor problems in the timber industry, which is taken to comprise logging and primary sawing. Among other things the meeting will deal with accident prevention and living conditions in logging camps.

—From *Health and Safety of Workers*, International Labor Organization.

Management and Union Health Programs

The Coordination of Care and Public Understanding and Acceptance of Health Services

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Changes in the needs and resources for health pose many problems of integration and coordination of services. Unless these problems are solved, the many forms of health protection and care available to and used by the members of a family are likely to be obtained with little regard to the health needs of that family and with slight if any attention to the economy and the efficiency with which the health personnel, equipment and facilities of the community are used. Moreover, costly gaps and duplications are likely to occur in the successive timing and use of preventive, diagnostic, curative and rehabilitative services.

The New York City Health Department has designed a study of the patterns and sources of health and medical services received by industrial workers and their families in New York City. The study will not only attempt, in a selected population, to analyze the types and volume of care received from all available sources of health services, but also to ascertain why one source of service was sought and used rather than another; why referrals were made for case finding, diagnosis, followup or treatment from one source to another; and what attitudes appear to motivate or guide past and probable future use of the services.

In preparing for the study a comprehensive list of references to sources of information on various types of health programs and services and their interrelationships has been assembled. In the belief that these references will be useful to many persons who may need prompt or frequent access to source materials on the origins, aims and operations of health programs in many parts of the United States, this bibliography is being published before the study in New York City is completed.

The bibliography is divided into five major sections:

I. Management Health Programs;

II. Union Health Activities;

III. Voluntary Health Insurance;

IV. Interrelationships Among Health Programs;

V. Public Understanding and Acceptance of Health Programs.

The first section, Management Health Programs, is presented here, and the four succeeding sections will appear in two future issues of *Industrial Medicine and Surgery*. The brief text which introduces each of these sections gives some comments relevant to the origin, development, current significance, or potential role of the type of program included in the section or to the importance of information available or being sought on the interrelationships and acceptability of the various programs within the community.

I. Management Health Programs

Management has had long and varied experience in making health and medical services available to employees. The earliest developments of these health programs were in frontier areas where medical services were unavailable.

When state workmen's compensation laws were enacted, the medical staff of the company frequently was responsible for care in compensation cases with the costs of such care met by the employer alone. Although the primary development of industrial health programs was closely related to the passage of compensation laws, the programs are currently recognized as having much broader values than those of first-aid and emergency service. The volume of absence from work because of nonoccupational ill-

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ness and injuries is nine times as great as absence due to occupational accidents and illness. To reduce sickness absenteeism industrial medical programs have therefore expanded their services to include care for illness and injury of nonoccupational origin. Added to their basic plans are such services as preplacement examinations, health counseling and education, diagnostic examinations and rehabilitation services.

Any substantial accomplishments in the expansion of these additional services will require the understanding and active participation of the general practitioner as well as of the officials of most of the voluntary health agencies, to say nothing of the acceptance and active backing of labor unions. When physicians in private practice and voluntary health agencies recognize that in-plant health programs will locate and refer to them many cases needing medical care, they probably will promote these programs more actively. As they observe the influence of the working environment on the health of their patients, and the part an in-plant program can play in the follow-up of patients, they also will be more willing to serve such programs on a part-time basis.

An indication of the widespread interest among the medical profession in industrial health programs is seen in the appointment of the Commission of Medical Care Plans by the Board of Trustees of the American Medical Association in November, 1954. In the progress report of the Commission covering the first year of its existence, the development of in-plant health programs and the expansion of their interest was described as follows:

"The increasing recognition on the part of management of the need to keep the employee on the job, to reduce absenteeism due to nonoccupational causes, to reduce turnover, to educate the worker in accident prevention and personal hygiene, to match the physical abilities of the applicant with the physical demands of the job—all have resulted in the expansion of what was originally conceived as a "first-aid" program into one of diagnostic and preventive care of "health maintenance," as it is popularly called today."

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Still a High Mortality

Based on 1956 experience, one out of every 100 boys and girls 14 to 19 years of age who are now entering the labor force will die as the result of a work injury. Six will suffer a permanent impairment and 70 will experience one or more disabling work injuries before reaching retirement. Only 23 out of the 100 will complete their working lives without a disabling work injury.

—From James P. Mitchell, Secretary of Labor, at "President's Conference on Occupational Safety."

XUM

Para Nitrobiphenyl,

A New Bladder Carcinogen in the Dog

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That para aminobiphenyl (PAB) (Xenylamine) is carcinogenic when it is fed to the dog was reported in April, 1954, by Walpole, Williams and Roberts, and in January, 1956, by Deichmann, Radomski, Anderson, Coplan, Woods and Heslin[2]. The final report of these investigators appeared in January, 1958[3]. Walpole's two beagle dogs developed advanced bladder tumors after ingestion of 30 and 34 g (2.9 to 3.3 g/kg) respectively, over a period of 33 months. The four mongrel dogs of Deichmann's group developed bladder tumors after ingestion of 87, 94, 129 and 144 g per dog (8.2, 8.4, 9.6, and 14.1 g/kg) over a period of 29, 21, 34, and 33 months, respectively. The first cases of human bladder tumors owing to PAB were reported by Melick, Escue, Naryka, Mezera and Wheeler[4] in December, 1955.

Para nitrobiphenyl (PNB)* was used as an intermediate in the manufacture of PAB. With the discontinuance of the manufacture of PAB in 1955, the use of PNB was likewise discontinued. Since commercial PAB was found to induce bladder tumors, and since the possibility of the presence of PNB as an impurity in the amino compound could not be overlooked, a study was undertaken to test the possible carcinogenic action of highly purified PNB in the dog. Obviously, bladder tumors noted in men exposed to Xenylamine may have been partially due to PNB.

Method

The investigation was initiated December 28, 1955. Four young adult female mongrel dogs, housed in a pen of seven by nine feet, were employed. They had no contact with other dogs. The animals were fed once a day at 9:00 AM. The ration consisted of wetted Purina Kibbled Meal and ground beef or horse meat. Hunt Club Bones (dog crackers) and water were supplied at all times. Para nitrobiphenyl (Eastman reagent grade) in a dose of 0.3 g was administered (in capsule) by mouth three times a week at 1:00 PM on Mondays, Wednesdays and Fridays throughout the experiment. Cystoscopic examinations were performed at intervals of five or six months.

Results

Table 1 presents the data. All dogs gained weight constantly throughout the 33-month experiment. Only one dog (D-37) displayed occasional mild hematuria following the biopsy after 25 months.

Dog D-37, long-haired, collie-type: Weight increased from 11.6 to 16.2 kg. Basal tumors protruding from many areas on the entire bladder floor beyond the trigone were noted on cystoscopic examination after 25 months, and after ingestion of a total of 98.2 g (7 g/kg) of PNB. Ad-

TABLE I.
EFFECTS OF FEEDING p-NITROBIPHENYL
(Each dog received three oral doses (by capsule) of 0.3 g PNB per week)

	Dog No. D-37		Dog No. D-38		Dog No. D-39		Dog No. D-43	
Start of Experiment	Dec. 28, 1955		Dec. 28, 1955		Dec. 28, 1955		Dec. 28, 1955	
Weight of Dog at Start	11.6 kg		17.5 kg		10.8 kg		10.8 kg	
Weight of Dog 33 Months Later	16.2 kg		24.7 kg		12.9 kg		13.7 kg	
Quantity of PNB Ingested When Tumors Appeared	g/dog	g/kg	g/dog	g/kg	g/dog	g/kg	g/dog	g/kg
1st Year	47.4	3.6	47.4	2.0	47.4	3.7	47.4	3.5
2nd Year	46.8	3.1	46.8	2.0	46.8	3.6	46.8	3.2
3rd Year	4.0	0.3	35.1	1.5	35.1	2.7	35.1	2.5
Total	98.2	7.0	129.3	5.5	129.3	10.0	129.3	9.2
Period of Treatment Before Appearance of Tumors	25 months		(negative at 33 months)		33 months		33 months	

*The carcinogenicity of this compound was announced at the 7th International Cancer Congress, London, England, July 6 to 12, 1958.

ministration of the compound was continued for an additional eight month period, at which time the dog was killed by electrocution.

Gross Examination: The spleen was bluish purple in color and slightly enlarged. There were several cysts in the mesentery of the pancreas, All other organs, except the urinary bladder, were without significant gross lesions.

The urinary bladder (Figure 1) showed irregular polypoid projections of the mucosa varying from 1 mm in diameter up to a larger tumor mass measuring approximately 2 cm in thickness. The large tumor mass showed some necrosis and ulceration on the surface. The cut surface of the tumor tissue was fairly firm, although friable, and grayish-white.

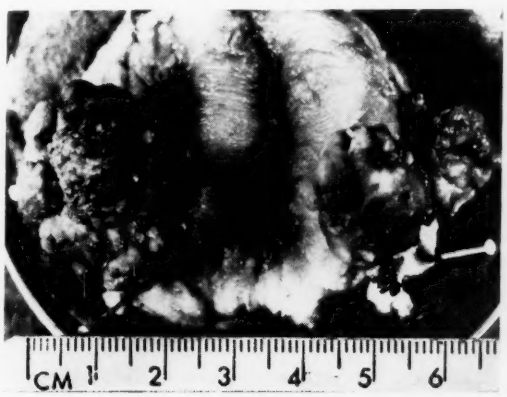

Figure 1.
Dog D-37: Urinary bladder showing small and large polypoid masses of tumor. The larger masses show some discoloration, necrosis and ulceration of their surface.

Microscopic Examination: Three sections of the bladder were examined and showed hyperplastic thickening of the mucosa with some tendency to squamoid metaplastic change. The superficial submucosa was quite densely infiltrated by lymphocytes and plasma cells, and in the deeper parts of the submucosa there were some larger lymphocytic accumulations with follicle formation.

The polypoid tumor masses (Figure 2) were composed of irregular masses of epithelial cells, often of squamous appearance but without keratinization or intercellular bridges. The cells were fairly uniform, and showed occasional mitoses. Infiltrative and invasive activity was evident at the base of the tumor, with irregular deep extension. The appearance was that of a carcinoma of the bladder with moderate anaplasia and a tendency to squamous change.

Dog D-38, German police dog type: Over a period of 33 months her weight increased from 17.5 to 24.7 kg. Cystoscopic examinations have remained negative. The animal has ingested a total of 129.3 g of PNB equivalent to 5.5 g/kg. Treatment is being continued.

Dog D-39, long-haired, chow type: The body weight of this animal increased from 10.8 to 12.9 kg. Bladder tumors were recognized after an experimental period of 33 months and after ingestion of 129.3 g of PNB (10.0 g/kg). The animal was sacrificed.

Gross Examination: The liver was coffee-brown in color and the edges were white and sharp. The spleen was bluish in color and slightly enlarged. The pancreas had white spots throughout. The kidneys were firm and had a rough surface. The capsule was difficult to strip from the surface. The heart showed a reddish

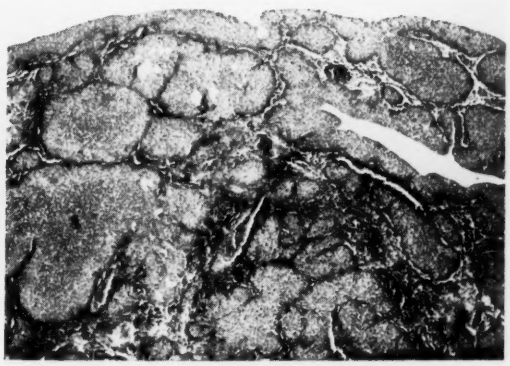

Figure 2.
Dog D-37: Polypoid tumor, composed of irregular nests and masses of neoplastic cells of squamous appearance.

bleb, approximately 0.5 mm in diameter, at the base of the mitral valve.

The urinary bladder showed several irregular polypoid projections of the mucosa, the largest of which was approximately 8 mm in diameter. The larger polyps were somewhat irregular on the mucosal surface and friable. The smaller nodules tended to have a smoother surface and were firmer. There appeared to be no deep extension.

Microscopic Examination (Figure 3): A section of the bladder showed focal areas of polypoid hyperplastic thickening of the mucosa, with squamous metaplasia. Enhanced growth activity with a moderate number of mitoses was evident in the thickened areas. The deep margin of one of the larger polyps showed some irregular downgrowth suggestive of early infiltrative activity, but at only one point did there appear to be definite invasion. The adjacent stroma was infiltrated by lymphocytes and plasma cells.

The appearance was that of focal hyperplasia of the bladder mucosa with squamous meta-

XUM

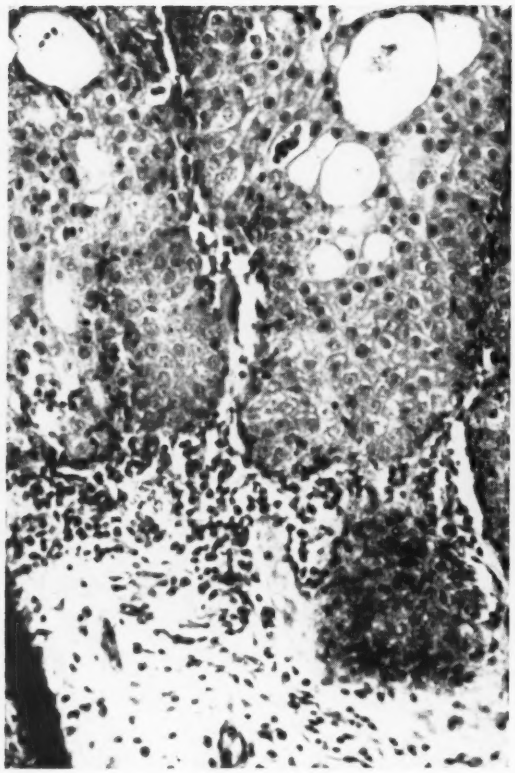

Figure 3.
Dog D-39: Urinary bladder, showing an area of hyperplastic polypoid squamous thickening of the mucosa, a focus of deeper invasion is shown, with more anaplasia, and mitoses.

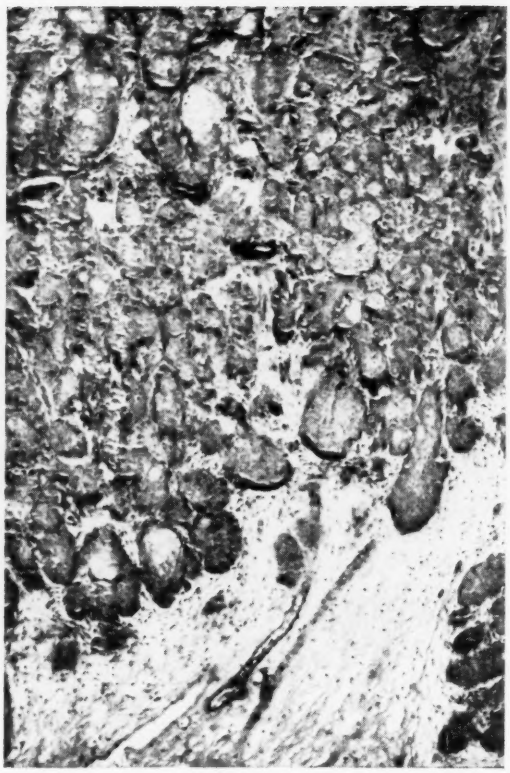

Figure 4.
Dog D-43: Urinary bladder, showing tumor composed of irregular nests and masses of neoplastic epithelial cells. There is irregular downward extension into the submucosa.

plasia, and focal polypoid epithelial tumor masses. One of the polypoid neoplasms appeared to have invasive growth characteristic of malignancy.

Dog D-43, long-haired, collie-chow type: This animal weighed 10.8 kg at the start of the feeding experiment and gained 2.9 kg during the succeeding 33 months. Tumors in the urinary bladder were noted after 33 months. The dog was killed.

The urinary bladder showed several irregular polypoid thickenings of the mucosa. The largest measured approximately 8 mm in thickness. One portion of the mucosa, 1.5 cm in diameter, appeared covered with a polypoid growth, the tissue being soft and somewhat friable. There appeared grossly to be extension of the growth into the submucosa, but not into the muscular wall of the bladder.

Microscopic Examination (Figure 4): Sections of the bladder showed polypoid mucosal tumor masses composed of nests and cords of anaplastic malignant epithelial cells. The tumor cells had predominantly a squamous appearance, but without evidence of keratinization. Lumen formation with a glandular appearance was present in some superficial areas. The neoplastic cells exhibited moderate growth activity. The tumor was extending deeply and irregularly into the submucosa. The adjacent stroma showed considerable infiltration by lymphocytes and plasma cells. The bladder mucosa away from the tumor area showed irregular hyperplastic polypoid thickening with squamous metaplasia.

The appearance was that of an infiltrating polypoid carcinoma of the bladder mucosa with squamous change, with hyperplasia and metaplasia of other areas of the mucosa of the bladder.

Comment

Comparison of the total doses and time interval required to produce bladder tumors in doses by para nitrobiphenyl and para aminobiphenyl are as follows:

PNB: 98 to 129 g/dog (7 to 10 g/kg) fed over period of 25 to 33 months.

PAB: 87 to 144 g/dog (8.2 to 14.1 g/kg) fed over period of 29 to 33 months[1].

It is obvious from these data that the PNB is approximately as potent a carcinogen as PAB.

Walpole, Williams and Roberts[5] have suggested that PAB may be metabolized to an ortho hydroxy amino compound and that it is this compond which is responsible for the production of bladder tumors. At this time, one can only speculate as to the metabolic compound or compounds that exert the carcinogenic action of PNB. It seems reasonable to assume that this compound is reduced in the body to PAB and that PAB is metabolized as previously discussed. If this assumption is correct, then it seems surprising that PNB should be as carcinogenic as PAB, for it is unlikely that the reduction is a quantitative one.

Summary

Bladder tumors were produced in mongrel dogs fed (by capsule) 0.3 g of para nitrobiphenyl (PNB) three times a week. In Dog D-37, tumors appeared after ingestion of 98.2 g (7 g/kg) over a period of 25 months. Dog D-39 demonstrated tumors after 33 months, and after ingestion of 129.3 g (10. g/kg) of PNB. Dog D-43 also ingested 129.3 g of PNB over the same period of time, but this dose was equivalent to 9.2 g/kg.

In these experiments with dogs, PNB appeared to be as potent a carcinogen as para aminobiphenyl (PAB) (Xenylamine).

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Proctoscopy in Industry

The employment of proctosigmoidoscopy as part of the routine physical examination of the 7,500,000 employees of business which have industrial medical departments would drastically reduce the incidence of rectal and colonic cancer, according to Thomas G. Rigney, M.D., senior physician, Standard Oil Company of New Jersey. Dr. Rigney and John R. Hill, M.D., section of proctology, Mayo Clinic, were the principal speakers at a conference in New York City on "Proctoscopy in Industry" called by the New York State Society of Industrial Medicine to discuss the feasibility of increasing the use of proctosigmoidoscopy by industrial physicians. The major objections to more general use of proctosigmoidoscopy as an office procedure, were patients' reluctance to preparation with conventional soap and water enemas and physicians' difficulties with patients who arrived improperly prepared for examination. Every one of the 4,000 Standard Oil employees covered by his department is offered a proctoscopy every one or two years, depending on age, physical condition and past history. Altogether, more than 6,000 such examinations have been performed, and approximately 10% of patients have been found to have adenomatous polyps. Consideration is being given to lowering the age for routine examination to 35 or even 30, he said, because of the higher mortality and lower five-year survival rate among younger patients with rectal and colonic cancer. The need for thorough rectal and colonic examination of patients generally was highlighted by Dr. Hill, who pointed out that 12% of all cancer occurs in this area, most of them arising from polyps "which must be treated as if they are potentially malignant." In 1955, 28,000 cases of cancer of the rectum alone were reported, and there were 11,040 deaths. It is imperative that every reasonable opportunity be taken to search for these lesions, since most of them give no warning signs. Approximately one-half of all malignant growths of the rectum and colon can be discovered on digital examination, and at least 70% are within reach of the 10-inch sigmoidoscope.

XUM

Immunization against

Influenza in Industry

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An employee immunization program is an example of the application of preventive medicine at its best. However, if reactions to the vaccine itself produce extensive absenteeism among the employees, the program is rendered considerably less effective.

For the past nine years, the Medical Department of Eastern Air Lines has conducted among its personnel a program aimed at the control of upper respiratory illness. This program has been kept on a voluntary basis and has been made available to all employees of Eastern Air Lines. The service consisted of the maintenance of a constant awareness of the principles of personal hygiene and of periodic immunizations. The educational phase emphasized to the employee the importance of avoiding obvious contacts, preventing chilling, and the wearing of protective clothing, even over perspiration-drenched clothes, when going into the cold weather. At the time of inoculation, employees were encouraged to purchase and use dietary supplemental vitamins and bacterial vaccine capsules. It is our belief that the purchase of such items is preferable to free distribution as our experience has shown that people are more likely to use drugs for which they have to pay.

Influenza virus vaccines have been employed throughout this program. The acceptance of vaccination against influenza nine years ago was low but has gradually increased to the point where now in the Miami base area there is about 40% acceptance and in the other stations about 30% acceptance. The difficulty with the outside stations is in getting the technical personnel to the people in small installations separated by great distances. The difficulty in Miami is the fact that in a subtropical area people tend to overlook the existence of cold weather with its associated respiratory illness. The acceptance of our program has been increasing about 10% a year.

With the recognition of the emergence of a new strain of influenza virus in 1957 arose the concern of the feasibility of controlling the illness caused by the new virus in military and industrial groups. It will be recalled that in the early weeks of the influenza pandemic there was a tremendous demand for, and a very small supply of, vaccine. It therefore appeared desirable to investigate the possibility of using smaller amounts of vaccine for the immunization of large groups of people. The existence of the preventive medicine program at Eastern Air Lines provided an ideal situation for trial of the new vaccine in a comparative study. Four hundred eighty individuals were selected for the preliminary study. These were divided into several groups subjected to different procedures, dosages and dosage schedules. These included intradermal injections of 10 CCA*, 20 CCA and 40 CCA, and subcutaneous injections of 100 CCA and 200 CCA.

Although the majority of the subjects received a single injection, some of the groups received repeat inoculations in 10 days or three weeks. Some of the individuals were given a late booster inoculation approximately three to four months after the initial injection. The serologic results obtained by mean of complement fixation and hemagglutination tests are presented in the ac-

*Chick cell agglutinating units.

Presented at Segunda Reunion de Medicina del Trabajo, Miami, Florida, August 18-22, 1958.

An extensive serologic evaluation entitled "Antibody Responses to Various Doses of Asian Influenza Vaccine in Children, Adults and Old People," by A. R. Beasley, M. M. Sigel, G. G. Schlaepfer, H. K. Edwards, F. M. Wellings, M. Mangels, Jr. and S. A. Gertman is presently in preparation for publication.

TABLE I.
FREQUENCY (%) OF SEROLOGIC RESPONSES TO SINGLE INJECTION
(Young and middle aged adults)

Vaccine	Route	Hemagglutination Inhibition			Complement Fixation		
		Positive	Doubtful	Negative	Positive	Doubtful	Negative
10 CCA	ID	27	34	39	43	7	33
20 CCA	ID	43	29	28	58	10	32
40 CCA	ID	65	25	10	35	50	15
100 CCA	SQ	86	8	7	77	3	20
200 CCA	SQ	63	11	26	81	3	16

TABLE II.
FREQUENCY (%) OF SEROLOGIC RESPONSES TO TWO INJECTIONS
(3½ - 4 WEEKS APART)
(Young and middle aged adults)

Initial Vaccine	Route	Second Vaccine	Route	Hemagglutination Inhibition			Complement Fixation		
				Positive	Doubtful	Negative	Positive	Doubtful	Negative
10 CCA	ID	20 CCA	ID	59	35	6	54	25	21
20 CCA	ID	20 CCA	ID	85	11	4	81	12	7
100 CCA	SQ	20 CCA	ID	100	0	0	76	9	15
200 CCA	SQ	20 CCA	ID	83	0	17	96	0	4

companying tables. Young and middle-aged adults from other industrial groups are also included in these tables. It may be seen that the response was the weakest with 10 CCA of antigen (Table I). A higher percentage of responses was found following injection of 20 or 40 CCA intradermally as measured by the hemagglutination-inhibition test. Second injections appeared to improve the antibody response, especially if they were given three weeks or later after the first. Table II shows the increased response in all groups following booster inoculations.

When planning the immunization on a large scale in industry, one should always weigh the eventual extent of protection against the possible inconvenience and reactions produced by the vaccine itself. Since influenza usually is a nonkilling disease and its major effect is loss of man-hours, it would be impractical to immunize against the disease if the immunization procedure itself were to provoke significant reactions among the personnel. It was therefore of interest to note that with two lots of vaccine, used in earlier studies, the extent of significant reactions (fever, aches, pains and chills) in individuals receiving 0.1 ml. of inoculum was about one-half that found in persons receiving 1. ml. subcutaneously. Approximately 5% of those receiving intradermal inoculation had significant reactions compared with 10% in the subcutaneous group. It must be kept in mind, however, that there was a variation between lots of vaccine, and with the vaccine of a third manufacturer, the intradermal injection did provoke significant systemic reactions to about 10% of the individuals.

Following this study, vaccine in 0.1 ml. amounts was offered to all employees.

Although the purpose of the study was primarily the evaluation of antibody response in the individuals receiving vaccine of various dosages and under different dosage schedules, the subsequent wide acceptance of the immunization procedures provided an opportunity to obtain some information about the vaccine's effectiveness. The figures provided by the Dade County Health Department showed the first major wave of influenza was during October and November of 1957, and the second wave in January, February and March of 1958. We do not have figures for the period represented by the first wave. However, the figures obtained during the months of December, January, February and March indicate three peaks of increased incidence of respiratory illness. If these three peaks are combined, and the incidence among individuals receiving vaccine and those nonvaccinated is compared, a significant difference is obtained in favor of those receiving one injection of vaccine.

The percentage of absenteeism due to respiratory illness during these three peaks among those receiving one injection of vaccine was 6.2 as compared with 17.1 among those receiving no vaccine. The breakdowns for the three periods are shown in Table III. Even when the absenteeism figures are narrowed down to only those absences of five days or more (more in keeping with the actual duration of illness due to influenza virus) the figures showed a difference in favor of the inoculated individuals (0.7% vs. 1.3%). These results suggest a protective effect of the vaccine administered in 0.1 ml. amounts. Furthermore, informal information received from station managers and chiefs of sections and departments indicates that Eastern Air Lines had more people at work during peak influenza periods than other industrial groups in immediate vicinities. Definite clinical conclusions, however, are not justified because the study was not designed along the lines of a well-planned statistical approach to the evaluation of protection. Furthermore, the total known over-all incidence of influenza in the Miami area, and especially at the Miami base of Eastern Air Lines, was low. This Medical Department of

TABLE III.
ABSENTEEISM

Number of Injections	Number of People	Dec. 16-22 No. and % Absent	Jan. 14-26 No. and % Absent	Feb. 17-March 3 No. and % Absent
1	2370	31 (1.3%)	53 (2.2%)	63 (2.7%)
0	1200	39 (3.3%)	71 (5.9%)	95 (7.9%)

XUM

Eastern Air Lines, in collaboration with the University of Miami School of Medicine, plans to continue long range evaluation of prophylactic measures for respiratory diseases, and at this time is preparing to organize a study in order to obtain more clinical information.

Summary

The Eastern Air Lines preventive medicine program aimed at respiratory diseases is briefly outlined. Serologic results obtained in a study employing Asian influenza vaccine are presented. They indicate antibody response varying in accordance with the amount of virus injected (lowest response with 10 CCA). The best responses were experienced following two injections. There were indications that the vaccine provided some protection as judged by the reduction in absenteeism owing to respiratory illnesses.

Shoe-Fitting Fluoroscopy

Since 1949 there has been increasing apprehension concerning the potential danger of radiation damage produced by shoe-fitting fluoroscopy. It was believed not only that this unnecessary exposure to x-rays could have an adverse effect on the hypophyseal development of children's feet but that many of the machines in use were inadequately shielded so that the store personnel operating them were receiving undue radiation. According to Kopp, the long interval between exposure and the occurrence of damage makes it difficult to exclude other etiological factors in which radiation injury may be suspected. However, the author describes a case of x-ray dermatitis of the foot in which there can be little doubt that the condition was caused by a shoe-fitting fluoroscope. In 1950-51 a few telangiectases occurred on the right great toe of a 56-year-old woman patient. The condition has since progressed and has spread across the dorsal surface of the foot, with atrophy of the skin, fine scaling and depigmentation. The patient sought no medical advice until 18 months prior to Kopp's report, when ulceration occurred beneath the nail of the right second toe, with no response to various local treatments. At this time there was induration of the soft tissue of both toes, and in the first three toes the nails were thickened and partially detached. Although the patient had allegedly never been exposed to x-ray examination or treatment, the clinical picture was one of typical x-ray dermatitis. Exploration of other possibilities of radiation exposure brought out the fact that the woman had worked for about 10 years in a shoe store in which fitting by fluoroscope was used. Both machines were tested and one was found to have defective lead shielding which exposed the operator to strong diffuse irradiation during fitting. It was found that the patient had in the course of years received very large doses of x-rays on the right foot. The woman operated the machine 15 to 20 times daily. Standing in the position normally occupied in operating the fluoroscope the feet were exposed only a little; however, if during the transillumination she supported her right foot on the platform in front of the shoe opening, it was exposed to intense irradiation. All shoe purchases were fitted by fluoroscopy, and if young children being fitted were afraid of the apparatus the patient often placed her own foot in the device to show that "it doesn't hurt." In addition, if there were no customers in the shop the salespeople occasionally tried new shoe models under transillumination. Kopp states that in this case the site and development of the x-ray dermatitis of the right foot indicates that it was caused chiefly by the exposure occurring while the foot rested upon the platform in front of the machine. The investigation of these machines showed the risk of damage not only to the foot but to the gonadal region as well. About 60,000 transilluminations per year were done with the two fluoroscopes, with an exposure time of from 30 to 45 seconds. During this period a nonscreened beam of rays passed from the front part of the foot platform to the direction of the customer's abdomen. The beam intensity in adults was approximately 1 r per minute and might have been much higher in children. According to Kopp, it is seen that a single defective shoe-fitting fluoroscope will raise the total radiation dose to the gonads much more than even very large radiological units. The author believes that, owing to the great danger involved by the use of shoe-fitting fluoroscopes, there is reason to investigate whether correct measuring and trying on do not fulfill the needs of exact fitting of shoes.

—From *What's New*, 1958, No. 206

Disaster Ambulance

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MOBILE FIRST AID STATION

During approximately 10 years of experience in refinery medical work, I have been impressed with the fact that many of the regular pieces of equipment are poorly suited to large industries. Specifically, the conventional ambulance is of minimal value in refinery medical operations. Admittedly, an effective safety program prevents frequent medical emergencies, but they can and do occur, and our experience has been that in such emergencies seldom is only one man the victim. Usually, two are involved, and we have had several experiences where as many as nine people have been simultaneously injured. Under such circumstances, makeshift ambulances have been pressed into use, but have been unsatisfactory. The equipment that can be carried in the usual ambulance is designed for conventional cases and ours seldom qualified in that category.

In 1955, our ambulance showed signs of advancing age, and it was decided to plan a replacement suitable to our needs. This replacement would be not only a multiple patient ambulance, but one that could also serve as a mobile first-aid unit. We had felt the need for this latter type of service on many occasions in the past, and our makeshift coverage in this field was far from satisfactory.

Our first planning called for considerable realignment of thought. Once we had gotten out of the habit of thinking in terms of conventional type of equipment, the entire medical staff was able to contribute many worthy ideas and suggestions. From these contributions and the discussions they engendered, came our present unit. It was ordered late in 1955, delivered in April, 1956, and put into operation late the same month.

The unit is designed to handle eight stretcher cases simultaneously. Oxygen is piped to the head of each bunk from a triple cylinder central source of supply. The cylinders are so manifolded that any one individually or all three simultaneously can feed into the system through reduction valves. The miniature resuscitator used directly on the patient is stored in clear plastic cabinets attached to the body panels at the head of each bunk. A suction unit, separate from the resuscitator operating on the oxygen system, is also stored there. The regular truck electrical system had a special generator added to provide the source of power for blinker lights, two-way radio,

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siren, backup lights and the clearance lights normal on such a vehicle. In addition to the regular heater, a large bus type heater was fitted into the rear compartment but operated through the engine electric system. Within the body itself, lighting is provided by a 110 volt system with a separate generator contained in the left rear compartment. This generator has its own self-starter and can be operated from the driver's compartment or from outside the vehicle. It provides 110 volt lighting through six fluorescent fixtures in the ceiling with a separate fuse box and switch. In addition, two inside and four outside 110 volt outlets were provided. This 3,500 watt generator can operate while the vehicle is in motion or standing still.

The vehicle is so designed that, having arrived at the scene of a disaster, it can function as a mobile first aid unit, permitting patients to enter through a wide rear opening and exit through the driver's compartment. The ceiling of the entire vehicle is raised to permit a six footer to move about easily without striking his head. One of the bunks can be cleared, and regular first aid supplies can be set up to provide a working area for the nurse serving the vehicle.

Our experience has shown that in the event of serious situations we are seldom at a loss for manpower, but are severely handicapped by our inability to control it. Communications under such situations are seldom satisfactory. To overcome this a loud speaker system with two fixed speakers on the front and two portable speakers have been provided. The microphone to this public address system is in the driver's compartment and orders to a group can be given and clearly understood more than 500 feet away. As a part of the public address system, a phonograph unit can be tied in on which "canned instructions," orders or announcements can be played. Portable flood lights can be worked in conjunction with this system. They are carried in a rear compartment and six outside outlets have been provided for them. This is particularly important as many of our night operations occur in remote areas of the plant where regular lighting is at a minimum. Other equipment which experience has indicated is most useful is portable resuscitator units with additional hosing and reduction valves so that they can be hooked into the main oxygen supply of the ambulance; three self contained "Chemox type" oxygen masks for the use of the ambulance crew; hand spotlights of the "sealed beam type" with 100 feet of line that can be plugged in through outside outlets.

The upper litter, rests and locks are designed to accommodate two Navy type Stokes baskets which we frequently use in our operations. Within the body proper, cabinets and drawers form the base for four aluminum stretchers on short legs. These stretchers are hinged for use in cardiac cases and are fitted with foam rubber mattresses which have removable outer rubber coverings. The cabinets provide storage space for extra blankets, aluminum safety helmets, heavy rubber boots, and one portion of the cabinet has been designed to contain a disposal unit for dressings and other excreta. All cabinet doors and drawers have been constructed to lock automatically when they are pushed into a closed position. They will not open even when turning a corner or putting the ambulance up at more than a 30° angle.

No heavy rescue equipment is included in this unit, as we are fortunate to have within the Medical Department a squad trained in heavy rescue work. Their own truck is designed to work in conjunction with our disaster ambulance.

Correspondence Committee

The Correspondence Committee on Occupational Safety and Health was formed in 1951 by amalgamating the former Correspondence Committees on Accident Prevention and Industrial Hygiene. The Committee now consists of about 170 experts, mostly specialists, from over 30 countries. Consultation with members usually takes place by correspondence, but from time to time panels have been convened for the drafting of technical studies, codes of regulations and other texts. One of the main tasks of the Committee in the future will be to collaborate with the Office in the preparation of an *Encyclopaedia of Occupational Health and Safety*. Up to the present two panels of the Committee have met, one in 1954 and one in 1955. The subjects discussed by the panels were planning of an *Encyclopaedia of Occupational Health and Safety;* the organization of industrial medical services; the substitution of harmless or less harmful substances for harmful substances in industrial processes; the preparation of an international list of notifiable occupational diseases; technical assistance in the field of occupational safety and health; revision of the *Model Code of Safety Regulations for Industrial Establishments for the Guidance of Governments and Industries;* classification and labelling of dangerous substances; prohibition of the sale and hire of inadequately guarded machinery and other industrial equipment.

—From *Safety and Health of Workers*, International Labor Organization.

Immunological Factors
In the Pathogenesis of

The Hyaline Tissue of Silicosis

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The histogenesis of silicotic nodules can be divided into two stages; a first stage of cellular proliferation and formation of reticular fibrils and a second stage of hyaline precipitation upon the fibrils. This second stage makes silicosis a disabling and progressive disease as compared with pneumoconioses caused by inert dust. Our research work on the origin of the hyaline stage of silicosis leads to the hypothesis that the hyaline tissue has an immunological origin and corresponds to an antigen-antibody precipitation. This opinion is supported by the following points:

1. The chemical composition of the hyaline tissue of silicosis has been subject to extensive studies in our Institute.[1,2,3] A summary of the results concerning the proteins, carbohydrates and lipids of the hyaline tissue of silicosis, compared with the hyaline tissue of spleen and the lung amyloid from a case of primary amiloidosis, is given in Table I.

From it the chemical similarities between the hyaline tissue of silicosis and amyloid, a substance for which the immunological origin is generally accepted,[4,5] are apparent.

2. The globulins which chemical (aminoacid) analysis identifies in the hyaline tissue of silicosis appear to be mostly β-globulins. They have been investigated immunologically by Pernis, Ceppellini and Ghezzi.[6] With this aim the hyaline tissue was accurately isolated from the pulmonary nodules of three cases of typical silicosis immediately after autopsy. The tissue was washed and homogenized in saline with a blade blendor. The resulting suspension was then washed and rehomogenized six times with saline to remove all traces of serum or lymph. The serum of a rabbit previously immunized with normal human serum was absorbed twice with one-third of its volume of homogenized and washed hyaline tissue of silicosis, and was then used as antiserum for immunoelectrophoresis (after the technique of Grabar and Williams).[7] The antigen used was a pooled normal human serum electrophoretically separated in an agar plate. As a control the same antiserum was absorbed with an equal amount of normal human lung treated in the same way.

The result of this experiment was the same in all the three cases studied. It is apparent that a single type of rabbit antibodies against human serum has been removed by absorption with the silicotic tissue, namely the type reacting with a protein fraction electrophoretically spread in the zones of β and γ globulins. No antibodies were removed by absorption with the normal lung tissue.

A similarly prepared homogenized and washed silicotic tissue as that used in absorption experiments was then injected intraperitoneally in rabbits, and antisera were obtained which reacted with normal human serum electrophoretically separated in an agar plate giving a single line of precipitation extending through β and γ globulins.

We can therefore assume that the acellular

TABLE I.
CHEMICAL COMPOSITION OF THE HYALINE TISSUES OF SILICOSIS AND PERISPLENIC SINECHIAE COMPARED WITH THAT OF LUNG AMYLOID FROM A CASE OF PRIMARY AMYLOIDOSIS
(The proteins are expressed as percentage of total proteins; carbohydrates and lipids are given in grams per 100 grams of protein)

Tissue	Proteins		Carbohydrates				Lipids	
	Collagen	Globulins	Hexuronic Acid	Hexos-amines	Hexoses	Methyl-pentoses	Total Lipids	Cholesterol
Hyaline tissue of silicosis	40.2	59.8	0.0	0.58	2.13	0.28	20.1	3.72
Hyaline tissue of perisplenic sinechiae	82.0	18.0	0.0	0.26	1.04	0.31	3.0	0.30
Lung Amyloid	41.2	58.8	0.0	1.22	2.20	0.36	14.3	3.60
Fibrinoid (Consden *et al.*)	70.0	30.0	0.0	0.90	3.60	—	—	—

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hyaline tissue of silicosis contains a globulin fraction which may be identified with a plasma-globulin having definite antigenic properties and the electrophoretic mobility of the β and γ globulins. It is interesting to note that Williams and Grabar[8] have shown that serum antibodies belong precisely to this fraction.

3. Some aspects of silicosis, such as the increased gamma-globulin content of the blood,[9,10,11] the proliferation of histiocytes and plasmacells in the reticuloendothelial system,[12,13] the progressive course of the disease, and the importance of the different reactivity of man for determining the onset and evolution of disease, all are features which are common to silicosis and to a wide group of diseases, the so called "collagen" diseases, for which an immunological origin has been demonstrated or appears very probable. Indeed the similarity between silicosis and "collagen" diseases is also borne out from what we have said under point one; as a matter of fact amyloid often appears in the course of many collagen diseases; moreover, on chemical investigation,[14] fibrinoid itself does not appear to be very far from amyloid or the hyaline tissue of silicosis.

One can say that practically the only major difference between silicosis and the "collagen" diseases is the localization of the process to the connective tissue of the lungs in the former case, as compared with the systemic diffusion in the other cases; this difference, however, is not difficult to explain if one admits (see further) that the autoantigens whose formation may be induced by quartz are not free to diffuse very far away from their site of origin. In this respect it may be interesting to consider the so-called Caplan's syndrome as the result of the mutual potentiation of two "collagen diseases", namely silicosis and rheumathoid arthritis.

4. The influence of tuberculosis in producing massive hyaline fibroses in silicotic patients can be explained on an immunological basis, assuming that the tubercle bacilli potentiate an antibody reaction, as it occurs when Freund's adjuvant (killed tubercle bacilli in a mixture of "aquaphot" and vaseline oil) is injected together with an antigen in experimental immunology. Indeed Freund's adjuvant has been particularly useful in potentiating the action of autoantigens.[15]

In order to develop further research along the lines of the working hypothesis which we have discussed it may be interesting to speculate on the possible mechanism by which quartz could induce the formation of antigens capable of starting an antibody response, and on the sites of formation of antibodies.

The main possibilities are the following:

a) Silica itself can act as an antigen.

b) Silica can induce the formation of autoantigens.

c) Silica acts as an adjuvant of heteroantigens.

d) Silica sets autoantigens free by killing a large number of cells.

This hypothesis must be discussed in the light of the limited knowledge we have at present.

First Hypothesis

A crystal of free silica might have antigen properties because of its particulate nature and its specific crystalline structure. However, the attempts of Claeys[16] to demonstrate antisilica antibodies in the serum of silicotic patients were not conclusive, and also researches by Antweiller[17] on the antigen properties of aerosil have given negative results.

Second Hypothesis

Silica could induce the production of autoantigens from proteins or polysaccharides.

Theoretically there could be three adsorption patterns of proteins on particulate matter:

a) simple adsorption,

b) adsorption with denaturation,

c) adsorption with denaturation and specific rearrangement of protein molecules after a particular model of crystalline lattice.

It is clear that the production of autoantigens would be most facilitated by the formation of proteins which were denatured following the third pattern, which is actually what would occur for proteins adsorbed on quartz according to Jäger[18] and Seifert.[19] Scheel and colleagues[20] have investigated to find out whether quartz denatures proteins in such a way as to form antigens and have been able to demonstrate that this in fact does happen following intravenous injections in the rabbit of quartz covered with homologous serum proteins; this does not occur with other dusts.

Holzapfel[21] recognised that certain carbohydrates, and particularly galactose, have a marked tendency to be adsorbed on quartz. The adsorption of certain tissue polysaccharides on silica crystals could give them autoantigenic properties. It is known that antigens and autoantigens (Cruikshank, Glynn and Holborow)[22] can consist of polysaccharides.

Third Hypothesis

The possibility that silica might act as an adjuvant of heteroantigens must be borne in mind in consideration of the fact that many finely divided dusts show adjuvant activity at a widely different degree, and also with a view to the fact that heteroantigens may easily reach the lung not only with the inhaled air but also with the blood.[23] Indeed this possibility might explain how some other dusts as kaolin whose adjuvant ac-

tivity is well known by the immunologist, may eventually induce a pulmonary fibrosis with formation of hyaline tissue which is morphologically and chemically indistinguishable from that of silicosis.

Lastly it is clear that the possibility that quartz acts at the same time as an adjuvant and as a source of (auto)antigens cannot be ruled out; that is to say that the second and the third hypothesis are not mutually incompatible.

Fourth Hypothesis

The possibility that quartz may start an autoantibody reaction by killing a large number of cells, is consistent with the knowledge that some substances which normally are kept inside the cellular membrane and do not come in contact with the circulating fluids may act as autoantigens when set free by cellular death or damage.[24]

As for the possible sources of the antibodies eventually involved in the silicotic process, we would say only a few words. First it is clear that the hystiocytes of the silicotic granulome might themselves be a first source of antibodies; secondly, the hylar lymph-nodes might similarly be considered able to produce antibodies, until they are totally invaded by the silicotic fibrosis. Moreover, if the autoantigens produced by the action of quartz were soluble, antibodies might be formed in different districts.

We consider that the immunological hypothesis of the pathogenesis of silicosis and of progressive massive fibrosis is a working hypothesis which opens a new and promising field of research.

The interesting point in this hypothesis is that it overrules the purely chemical or physical theories on the genesis of silicosis and emphasizes the role played by the response of the organism to the action of silica. The attention is focused more on the reaction of tissues and of the whole organism than on the toxic or irritating action of the foreign body.

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Encyclopedia of Occupational Health and Safety

An encyclopedia entitled Occupation and Health, compiled by the Office with the help of members of the former Correspondence Committee on Industrial Hygiene and nearly one hundred collaborators, was published over the period 1930-1934. It consisted of two volumes, containing 410 articles and totalling 2,300 pages. Supplements were published from 1938 to 1940. The encyclopedia is now out of print and will be replaced by a publication of broader scope to be called Encyclopedia of Occupational Health and Safety. It is now in preparation. Up to the end of the war the Office published two series of technical studies, one on safety topics and one on health topics. These series have been discontinued and will be replaced by the Encyclopedia. From 1925 to 1950 safety news of international interest was published in Industrial Safety Survey which appeared every two months up to 1939 and quarterly from 1940 to 1950. In 1951 its scope was broadened to include occupational health as well as occupational safety and its title was changed to Occupational Safety and Health. Each issue comprises (1) one or more leading articles, usually by an expert outside the Office; (2) a section entitled "Reports and Activities", which includes summaries of factory inspection, mining inspection and similar regular Government reports, summaries of special reports on particular subjects by official or semi-official bodies, and notes on safety and health institutions and kindred bodies all over the world; (3) summaries of texts of new laws, regulations, codes, standards and the like; (4) reviews of recent books and articles in periodicals; and (5) reproductions of new posters issued in different countries. It is published quarterly in English, French and Spanish editions. —From *Safety and Health of Workers*, International Labor Organization.

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Profiles in Occupational Health

Andrew Meiklejohn

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I first met Andrew Meiklejohn 22 years ago on the day that we both became members of the Association of Industrial Medical Officers. I was puzzled by the apparent meekness of this rotund and sturdy Scot with his firm jaw and piercing eyes. Scant of hair tinged with a reddish hue, he looked more like a Viking warrior than an industrial medical officer, and to a young man who had recently qualified in a London medical school, such humility in a doctor was strange. I remained puzzled until I heard him castigate an unfortunate friend who spoke with levity about injuries to the hands. Dr. Meiklejohn reminded us that a workman's hands were his only capital and an injury could lead to manual bankruptcy. He has also been known to flay those who think of diluting their Hippocratic oath in the laboratory of industry. To him "industrial medicine is basically a personal service to the individual workman and so cumulatively to the group".

And in time by knowing him, I learned the truth of King Harry's words to his soldiers at Harfleur,

"In peace, there's nothing so becomes a man
As modest stillness and humility.
But when the blast of war blows in our ears
Then imitate the action of a tiger."

Wounded in the leg at ill-famed Armentiére in World War I, Andrew Meiklejohn had the choice of being rehabilitated in the Army Pay Corps or medical school. Guided by his native instinct and with an unusual lack of interest in pay, he hobbled on crutches to Glasgow University to be a medical student. He qualified with commendation in 1923. (He will admit only to uneventful progress.)

Like so many bright Scottish stars, he followed the track to the south, eventually landing in Manchester as a Tuberculosis Officer. Here, he was destined to change course into the orbit of industrial medicine. To find an outlet for his abundant energy, he attended the public health lectures at Manchester University where he was deeply moved by the teaching of Sir Thomas Legge. His own words testify,

"As he (Legge) walked before the benches, one felt he was pacing the factory or foundry floor exchanging experiences with the workmen . . . Our notebooks pushed aside, we moved with him from factory to factory . . . And all the while he excited in us something of the beauty of craftsmanship. By any standard — professional skill, scholarship, social purpose and, above all, sympathy and integrity, Legge was a great man."[1]

In 1930, Legge persuaded Meiklejohn to join the Silicosis Medical Board and on June 1, 1931, Meiklejohn was sent to Stoke, the heart of the English pottery industry. His arrival in these black pottery towns coincided with the advent of a new law demanding the periodic examination of pottery workers exposed to a risk of silicosis. He was able to study the effects, on the incidence of silicosis, of using alumina instead of the dangerous flint powder in the china warehouses. The success which followed this simple metamorphosis of danger into safety belongs to the industry and to the men whose lives were prolonged by it. But they all owe much to Meiklejohn who inspired employers and workmen to a common purpose of prevention. In 1946, he was repatriated to Glasgow where he hoisted the flag of industrial medicine in the University and has flown it with candor and scholarship, without hot air and humbug.

He has pursued his historical researches of men and diseases of industry. One of his most notable contributions is a recent biography of

Charles Turner Thackrah,[2] a turbulent Sassenach from Leeds who pioneered industrial medicine in the nineteenth century. Meiklejohn has frequently left his beloved Glasgow to invade England, to see his old love — the potteries, to be President of the British Association of Industrial Medical Officers or just to follow Robert Burns,

"Over the border
And gie them a brush;
There's somebody there
We'll teach better behavior".

At times submerged by illness, he has bobbed up like a cork with renewed vigor. Two or more years ago, he was clutched back to life all but dead of an internal hemorrhage. Since then, he says, he has lived in borrowed time; this he is repaying with an interest compounded of humor and humanity.

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Is Preventive Medicine a Menace?

According to Hanlon, public health measures, by making possible longer healthier lives for more people, have been called imprudent, irresponsible and even immoral. It is true that changes, especially if they are sudden, bring with them certain dislocations, but — let's face it — change is inevitable and if properly directed should result in benefits to mankind. Preventive medicine is censured on the ground that it has caused so explosive an increase in population as to make it impossible to feed and house all the people now living; and those who are forced into a low standard of living are the ones chiefly responsible for crime. Public health measures have indeed made important contributions to the increase in population that has become so evident. They have done this by improving chances of fruitful conception in young married couples, increasing the chances of survival for the infant and preventing death in young adults, thereby reducing the number of marriages dissolved by death. To attribute increase in population solely to public health, is, however, erroneous because the present increase began about 1650 or before modern public health was known. Because an industrial civilization can support more persons per square mile than an agricultural civilization, the industrial revolution saw a population upsurge. Any factor that raises the standard of living and improves conditions of work brings an increase in population in its wake. Such changes may be based on changes in government, social reforms and in employer-employee relationships as well as disease prevention. The disturbances in equilibrium caused by sudden increases in population are temporary because improved standards of living and area saturation are always attended by a decrease in the natural birth rate even when no measures to control conception are widely practiced. The dramatic cutting of the death rate in certain backward regions is chiefly due to the application of modern methods of controlling communicable disease. This will not continue indefinitely but will quickly level off as the life span increases and the death rate from degenerative and other diseases of later life increases. The factors that cause a decrease in the death rate from communicable disease do not cause an increase in the per capita birth rate but may even cause it to decrease. It would seem reasonable to conclude that public health measures have been criticized largely because their effectiveness in some regions has been so dramatic. We must convince the detractors of these measures that man's future is to be found not in a continuation of sickness and misery but through health and enlightenment. It is clear that closer study of the relationship between population and public health and the interrelationship between public health and other factors affecting population is needed. The problem is not how to curtail health measures but how to gear production, distribution, education and peaceful cooperation between nations to keep pace with the advances in disease prevention.

—From editorial in *World Medical Journal*, July, 1957.

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Alcoholism: *The Concern Of Industry and the Community*

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Alcoholism is truly designated as a disease today by the American Medical Association, the U. S. Public Health Service and all leading medical groups. It is indeed a disease and its implications are many. It is a sociomedical problem which involves more than the physical condition of the victim. It invades the total personality of the individual who is affected pathologically, emotionally and spiritually. It also affects the society in which he lives, his family and the persons with whom he associates at work or in business.

Just as alcoholism invades the total personality of the individual afflicted with it, so the results of this condition, in the aggregate, affect the total community. The police courts, the social agencies, the pastor in his study, the doctor in his office and the industries of the community all are inevitably touched by it. All groups making up part of the community, therefore, should be concerned about it, and industry is, of course, a leading part of almost every community.

It is important to remember that attitudes around the victim of an illness have a direct bearing on his recovery potential. The attitudes of the people of his community influence not only his own conception of his problem, but also his all-important decision of whether to do something about it. If the alcoholic is considered to be one thing by his plant manager or the medical department of his company, and another thing by the social worker or by his minister or priest, it is going to be difficult for the patient to believe and accept the true nature of his condition. All segments of the community should be operating on the same "wave length," as it were.

It was in order to bring about community understanding, a change of attitude and a new climate of understanding, as well as to unify all segments of community thinking on the problem of alcoholism that the National Council on Alcoholism was established some 14 years ago as the national voluntary agency in this field. The procedure was exactly the same as that in the other fields of health such as cancer, tuberculosis, mental health and heart disease. The purpose, of course, is to bring understanding to the community with the ultimate goal of prevention and reduction of this disease, which is now ranked number three in prevalence and probably should be considered first in its economic waste and costliness.

No community attack on a major problem of health and welfare can be undertaken without consideration immediately being given to industry's role. Industry is a vital force in every community today and, as we all know, has played an admirable and responsible role in helping to meet the needs of communities in which it is involved. I am not intimating in any way that industry is to be considered solely responsible or totally charged with the entire attack upon this major problem. Industry, like other segments of the community should and, of course, will bear its share of responsibility for dealing with it, but I fear that too often it has been led to feel that it is not only responsible for causing a great portion of the incidence of alcoholism in a community, but that it should accept a major and disproportionate role in dealing with it. It has never been the belief of the National Council that industry should be asked to accept more than its share of responsibility for the alleviation and treatment of this condition, any more than for any other community problem.

But just as in the case of any other major problem, industry does have a stake in cooperating with community efforts in the attack on alcoholism, and I am happy to say that it has begun most definitely to indicate a desire to take action. This has been shown by the growing number of corporations which have established their own programs for early detection and treatment of alcoholic employees on a basis of treatment and rehabilitation as opposed to the outmoded procedure of firing. Again it has shown it by its support of community programs — not only financial support, although this has been vital in many areas, but also by the leadership it has given through many individuals in top management who have served on the board of local community programs known as Committees on Alcoholism.

The National Council on Alcoholism's approach has been to pinpoint the attack on alcoholism through the communities, under leadership well-versed in the local situation, and that knows the existing potential for meeting the need and recognizes the importance and incidence of the problem. The National Council attempts to guide and support local committee programs of education, which result in the development of treatment resources. Thus, if industry does aid in this community approach, facilities and resources

Presented at the Industrial Medical Conference, Symposium on Alcohol, Atlantic City, New Jersey, April 22, 1958.

come into being which it can utilize for its employees and which give both the community and its industries a well-rounded choice of inpatient, outpatient, professional and therapeutic resources for diagnosis and treatment.

The needs of each alcoholic vary. Therefore, the needs of alcoholic employees will vary. There are numerous resources which can be developed in a community and which exist in many places today. First of all are the groups of Alcoholics Anonymous, known to all at least by name, and which exist in almost every city, town and hamlet in the country. The Alcoholics Anonymous therapy has perhaps been responsible for more recoveries from this disease than any other method. All alcoholics, unfortunately, do not respond with equal success even among these effective groups, and this is primarily because many persons with alcoholism have more deep-rooted and neurotic factors involved in their problem than do others. Therefore, some form of psychotherapy and psychological counseling is frequently necessary (and often this is done when the patient is also going to AA). In recent years, the alcoholic clinic has come into being where, on an outpatient basis, through treatment by a clinic team, the deeper needs of the alcoholic are encountered, diagnosed and treated. Long-term psychiatry is sometimes needed after diagnosis and screening in the clinic, and psychiatrists in a community should be available privately for this type of therapy. In the acute stage of his illness, the alcoholic is in need of hospital care. Many hospitals even today will not accept alcoholics in the acute condition and, although this condition rapidly is being remedied, there is a need for more effort by community leaders to gain acceptance for this type of patient in local hospitals.

A number of general practitioners should be available in any community for the medical management of the alcoholic who does not need hospitalization, but who does need medication and care prior to the long-term therapy which is the basic treatment for the alcoholic. Also, many private practitioners, clinicians and treatment centers conduct group psychotherapy sessions, where, instead of on an individual basis by a psychiatrist, this type of treatment can be given in a group at less expense (and which also presents, for many alcoholics, a more favorable setting).

It is the task of a community program to develop these resources if they do not exist. I am happy to say that they do exist in many major cities, and have been developed through community action with industrialists and industry playing a major role. Where these have come into being through community action and leadership, industries are using these resources. Many companies in Worcester, Massachusetts, use the St. Vincent's Clinic as well as the Alcoholism Information Center for referral from their medical department. In Rochester, New York, similar resources are available. In Houston, three outpatient clinics, inpatient hospital care and an Alcoholism Information Center are all available to local companies and branches of national corporations. In Detroit, the outpatient alcoholism clinic and the Information and Consultation Center of the Detroit Committee on Alcoholism came into being through industrialists getting together and insisting that such a facility be established with funds provided by the United Fund.

In Corning, New York, members of top management of the Corning Glass Works and Ingersoll-Rand Corporation decided that before putting in their own in-plant programs of education of supervisors and foremen as well as a plan for helping alcoholics in the medical division, the first step should be establishment of community resources. It was felt that it would do little good to have the company medical division and personnel department fully informed on this subject and giving sound information and counsel to its employees if the latter were obliged to go out to an uninformed community and receive contrary advice from social workers, physicians, clergy, etc. After one year's effort on the part of the Corning Area Committee on Alcoholism, these companies have instigated their own in-plant educational programs for early detection, and with the cooperation of employees, they are now referring their patients for long-term treatment to community resources.

Perhaps some of you are not aware that such resources may exist in your community. If not, the National Council can send you a list of the location of its committees and also the annual National Directory of outpatient clinics.

Even if there is nothing in your community, it is possible that your company could take or share the lead in bringing together leading citizens who might wish to develop such a program. At least, any industry can have an enlightened medical department with a staff well oriented to the nature of the alcoholism problem, able to use modern techniques of diagnosis, and able to give initial counseling and medical care to meet the basic needs of the problem drinker.

We who work full time with the alcoholic program are most grateful to the industrial medical directors who have often shown more enlightenment than management concerning the magnitude of this problem in industry and its concomitant cost in loss of manpower. These medical directors have helped establish inplant understanding so that employees can come to them for discussion of their problem without fear.

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Letters

Compensation — Historical Note

We have noted with interest an editorial appearing in the July issue of *Industrial Medicine and Surgery* questioning 1958 as the fiftieth anniversary of a significant milestone in workmen's compensation legislation. We think there is some confusion as to the identity of the Federal law whose anniversary date is being observed this year. In 1908, Congress passed two separate laws involving protection for injured workers in the course of employment. One law, commonly termed the Federal Employers' Liability Act, was enacted on April 22 (ch. 149, 35 Stat. 65) and was concerned with the liability of common carriers engaged in interstate commerce for injuries suffered on the job. This act provided for abolition or modification of the common-law defenses that could be pleaded by railroad employers in damage suits instituted by injured workers or their survivors. If this were the legislation whose anniversary is being observed this year, then we would be in agreement with the editorial as to its relative insignificance.

However, the second law, commonly known as the Federal Employees' Compensation Act, enacted on May 30, 1908 (ch. 236, 35 Stat. 556), provided for monetary payments to certain Federal civilian employees engaged in hazardous employment or their survivors when injured or killed on the job. While limited in scope, this law did embody the workmen's compensation principle that a worker incurring an occupational injury should be compensated according to a regular scale of payments —regardless of fault and without the necessity of court action. It is this second law to which writers in the field have reference when noting the fiftieth anniversary of workmen's compensation.

As you point out, six years earlier, in 1902, the State of Maryland had enacted a workmen's compensation law, but this law was declared unconstitutional. It is therefore believed that the Federal Employees' Compensation Act of 1908 can properly be termed the first *effective* workmen's compensation law in the United States.

A recent study, for instance, points out that "The first major impetus to American legislative action came in 1908 when, under President Theodore Roosevelt's leadership, Congress enacted the first effective American compensation law, covering civil employees of the Federal government." (Herman M. Somers and Anne R. Somers, *Workmen's Compensation*, New York, 1954, page 30.) An earlier study makes the point that this law "had no little effect on the workmen's compensation movement, particularly in view of the distinct recommendations in the President's message that the principle proposed . . . should be made applicable to employment generally." (Clarence W. Hobbs, *Workmen's Compensation Insurance*, Second edition, New York, 1939, page 89.) Another writer comments "Although limited in scope, niggardly in benefits, and crudely drafted, it was nevertheless the first real American workmen's compensation law." (Domenico Gagliardo, *American Social Insurance*, Second edition, New York, 1955, page 388.)

The first *State* workmen's compensation legislation to stand up in court were those laws enacted in 1911 by about 10 States, with Washington and Kansas having the earliest enactment dates and Wisconsin the earliest effective date. If it is desired to observe the fiftieth anniversary of state workmen's compensation legislation, then 1961 would be an appropriate year.

—IDA C. MERRIAM
Director, Division of Program Research
Department of Health, Education and Welfare
Washington, D.C.

Editor's note:
Readers concerned with workmen's compensation will be interested in the bibliographic reference to the recent *Social Security Bulletin*, of Department of Health, Education and Welfare, Social Security Administration, Washington 25, D.C.

Books

Industrial Psychiatry

MENTAL HEALTH IN INDUSTRY: A. A. McLEAN AND G. C. TAYLOR. McGraw-Hill Book Co., Inc., New York, 1958, pp. 262, $6.50.

Among those engaged in student training in occupational health is the complaint that text material on mental health in industry is scanty. More directly the complaint is that authors highly skilled in psychiatry and psychology all too often display little familiarity with industry itself. The contents of some such books might be equally applicable to mental health in military establishments or in gerontology. In contrast, this present book has the ring of closeness to industry.

The authors themselves must have been aware of the need for closeness between the psychiatrists and the work place when they wrote:

"The psychiatrist in industry should possess certain qualities which will enable him to be an effective person, to do a good job, and be accepted by everyone in the company from the janitor to the president. He must be accepted first as a person, before he can be accepted as a psychiatrist. The employees should feel that he can understand them and that they can understand him. He should be humble, especially in the use of his language, which should be simple and understandable. His ability to get along with his colleagues must be unquestioned. He should have the adaptability to work within the framework of the organization, remembering constantly that there is always company policy which must be considered before he decides to act. He must be able to work with groups, therefore his ability to mix easily should be pronounced. Conferences will consume much of his time. The psychiatrist's ability to confer graciously and effectively with his superiors, subordinates and all others, both professionally and nonprofessionally, is a test of his adjustment flexibility. In short, he should be a mature, tolerant person who understands both himself and other human beings."

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The Changing American Family and the Woman Labor Pool

The Health Information Foundation, through its "Progress in Health Services" (volume 7, number 7, September, 1958), reports upon an investigation concerned with health and the changing American family. From a first look it might appear that all such is remote from occupational health. A closer look promptly establishes many areas of closeness. At all times industry's demand for labor determines the number of workers, but not necessarily the ratio between men and women employees. The number and percentage of women workers in industry oftentimes are influenced by changes in family life—the divorce rate, the rate of widowhood, the number of children in the family, the age at marriage, etc. On these accounts, the following excerpts from this report become pertinent.

Improvements in health promote family stability, and family life seems to promote health. Married persons in this country have a substantially better health record than the unmarried. Moreover, the relative advantages of the married are now greater than formerly, especially for women. Today even childbearing — once so dangerous — does not raise the mortality of married women above that of others through the reproductive period or beyond.

In 1900, 27.4 marriages in each 1,000 ended each year in the death of one spouse (in a small proportion of cases, the simultaneous death of both). The rate declined to 24.8 per 1,000 in 1924-28. Thereafter the drop was much sharper, and by 1957 it was only 16.6 per 1,000, a level about 40% lower than at the turn of the century.

Currently, about 750,000 marriages are broken by the death of a husband or a wife each year. (In more than two-thirds of the cases, by death of a husband.) If the rate had not declined since 1900, as many as an additional 400,000 marriages might now annually be dissolved by death.

Moreover, dissolutions by death now occur at older ages and after a longer average duration of marriage. The median age at death for married persons was 53.6 years in 1900, and 56.9 years in 1924-28. By 1949-51 it had reached 63.2, and in 1957 it was 65.2 — a gain of 11.6 years since 1900. As for duration of marriage, white persons entering their first marriage in 1900 could expect, on the average, about 30 years of married life together before one of them died. By 1956 this figure had climbed to 43, a gain of about 13 years.

The decline in marital dissolutions by death has been partly responsible for the fact that a much larger proportion of Americans are now married. Among the population aged 14 and over in 1957, 67% of the men and 66% of the women were married; in 1900 the proportion was 57% for either sex.

Another result of the decline in marital dissolutions has been to move widowhood to the older ages. The median age of all widowed persons of either sex rose by 10 years from 1900 to

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1957, from 57.8 to 67.8. The rise was particularly rapid after 1920. In 1900, young widowed persons (under age 45) constituted nearly one-fourth of all widowed, but by 1957 the proportion had dropped to just over 6%. During the same period, the proportion of widowed at the upper ages (65 and over) increased from one-third to nearly three-fifths of the total.

Since the decline in mortality has been sharper among women than among men, the average age at death is now higher among women. As a result of this — and because wives are usually younger than husbands — the average age at which men become widowers tends to run somewhat higher than the age at which women become widows. While in 1900 the median ages of widows and widowers in this country were exactly the same — 57.8 years — by 1957 the average widower was 4.3 years older; the median ages were 71.1 for widowers but 66.8 for widows.

There are now just under 10 million widowed persons in this country, and the number is increasing rapidly. Currently about three-fourths of the total are women. Since 1950 widows have been increasing by about 115,000 annually, while the number of widowers has actually decreased somewhat.

Today the widowed comprise 3.8% of the male population and 12.6% of the female. At the young ages their proportions to the total are very low, but they increase rapidly as age rises. At 75 and over the widowed make up 38% of the male population and 70% of the female. The proportions of widowed to total population are higher among the nonwhite population than among the white.

Board Diplomates

A recent issue of the *Journal of the American Medical Association* (October 4, 1958) extensively is devoted to American Medical Specialty Boards. One section reports the total number of occupational medicine certifications. Three hundred forty-seven certifications have been awarded as of June 30, 1958. However, it is unlikely that this number includes the candidates who successfully passed the examination following the Atlantic City meeting in April. The formality of inducting that group awaits the session of the American Board of Preventive Medicine sitting in St. Louis at the time of the American Public Health Association meeting. It is anticipated that from 50 to 75 additional certificates will be awarded at that time.

The total membership of the Industrial Medical Association approximates 3,500. By coincidence this published figure of 347 occupational medicine diplomates constitutes almost an exact 10% of the total membership. In the years just prior to the creation of the occupational medicine section of the American Board of Preventive Medicine more than one dissident scoffingly predicted that the industrial physicians would prove to be so liberal that no less than 50% of the total membership of the Association readily would become diplomates. Actual developments distinctly to the contrary mark this Certified Medical Specialty group as representing one of the lowest in relation to over-all number of physicians to be considered. It is well known that the requirements for certification by the American Board of Preventive Medicine are indeed stringent and the examinations imposed are definitely difficult. All considered, the attainment to certification by this American Board of Preventive Medicine more and more is becoming a prize distinction.

Yet it must be apparent that from the total membership of the Industrial Medical Association (3,500) there must be far more than 347 fully worthy of recognition as a certified specialist. It is not a prospect that the requirements for certification will be lessened in foreseeable years. In fact, the requirements may become more rigid. In order that this number of diplomates in occupational medicine may in truth include every deserving industrial physician, all such are referred to the Board requirements as set forth in that issue of the Journal of the American Medical Association above indicated. It may be assumed that the next examination will be arranged to coincide with the date and place of the annual conference of the Industrial Medical Association in 1959. It is to be hoped that the number of candidates seeking certification through the examination procedure may be higher than at any prior time.

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*Deceased

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